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LXV.

CORRELATIONS BETWEEN THE ACUITY FOR HEARING AIR AND BONE TRANSMITTED SOUNDS IN RINNE NEGATIVE AND RINNE POSITIVE CASES.*

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ST. LOUIS.

It is well known that air sounds may be transmitted to the cochlea either by way of the drum membrane and ossicular chain or directly through the medial tympanic wall. It is quite universally accepted that the former route is the more efficient one. If it were possible to test a normal individual before and after removal of the drum membrane and the two outer ossicles, then the difference in the intensity required under the two conditions would establish the relative efficiency of the two transmission routes. And if it was definitely shown that the one pathway was

*This paper was presented before the St. Louis Ear, Nose and Throat Club, March 10, 1930. The reader is referred to an article by Sonntag (Arch. f. Ohren-Nasen- und Kehlkopfkunde, Vol. 125, March, 1930, p. 79), on "Das Klinische Wert des Oto-Audion," where a similar series of experiments are reported and with strikingly similar results.

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far more efficient than the other, then the probabilities of a simultaneous direct and indirect contribution to the intensity reported might also be determined. Inasmuch as this experimental evidence cannot be obtained, the information must be arrived at in another manner.

Similarly, bone sounds may be transmitted to the cochlea by a direct transmission of the vibrations through the wall of the otic capsule, or indirectly by way of the drum membrane and ossicular chain. Here the problem of the relative efficiency in the two possible routes is still further complicated because while air sounds may be exhibited to either ear, bone sounds tend to affect both labyrinths. Indeed, this fact is the basis for one type of lateralization. No method has been discovered thus far which makes a test on either ear possible through bone transmission.

A simultaneous registration of both air and bone sounds may occur, although this condition probably obtains for the most part where the sound production lies within the individual. It explains why one fails to recognize the phonographic reproduction of his own voice, and also the subjective changes in voice quality on occlusion of the external auditory canals or where the drum membrane is loaded with secretion or pus.

It is, therefore, important that the available experimental evidence on the correlations between the air and bone acuity be submitted because such correlations are useful in the differential diagnosis of auditory disabilities. This paper is a continuation of the quantitative studies by Kranz and myself at Riverbank Laboratories, and was completed at the audiometric laboratory of Dr. John F. Fairbairn of Buffalo, N. Y.

The Rinne test is a particularly important one because a direct comparison is made between the acuity for hearing the air and the bone sounds developed by a vibrating fork. Unfortunately, the method has obvious limitations from both a physical and a physiologic point of view and these need not be discussed here. A direct comparison of air and bone acuity, however, is also possible by means of audiometric methods which overcome, in part, the objections to the fork test. The air and the bone sounds may be delivered by an air and a bone receiver, respectively,

under definite quantitative control and throughout the entire frequency range.

All tests reported here were made with the same apparatus and under the same conditions. A beat frequency oscillator was employed as a source and the attenuation of the signal was accomplished by means of an audibility meter connected to a standard receiver. The bone acuity was established by applying the bone receiver, described by Kranz and myself,[1] to the forehead. It was found necessary to amplify the oscillator output for the bone telephone and this was done with a Western Electric "7a" amplifier. The oscillator was always tuned to 1024 Hertz at 26 on the variable condenser dial, and this yielded a frequency range of about 120 Hertz at 5 to about 8000 Hertz at 75—the limits of the range tested. The base line of the figures is spaced off in terms of each five points on the condenser dial and the "C" frequency values have been ruled through for purposes of pitch orientation. The attenuation of the signal is represented by the steps on the audibility meter, where "0" represents the maximum output and "32" the minimum output. The higher the curve, therefore, the greater the sensitivity. The air acuity curves are plotted in steps of two; while the bone curves were plotted in single steps. The apparent correlations between the two types of curves may, therefore, be considered quite empirical and somewhat accidental.

One hundred cases were selected out of some three hundred studied. The selection was on the basis of a more less uniformly bilateral disability, as shown in the air acuity curves, and only those cases were chosen in which there was no obvious loading of the drum membrane with secretion or pus. The cases were next divided into two general classes: Rinne positive and Rinne negative. Each Rinne positive and Rinne negative class was further subdivided into three groups on the basis of the upper frequency limit heard through the bone telephone at maximum output. Group A, made up of sixteen normal individuals (Rinne positive) as a control, and forty-seven Rinne negative cases, heard the bone telephone to the upper limit of the test. Group B, composed of fifteen Rinne positive and fifteen Rinne negative cases, did not hear the bone telephone above 50—60 (roughly about

4000 Hertz). Group C includes fifteen Rinne positives and eight Rinne negatives who did not hear the bone telephone above 40 (2048 Hertz). The cases were averaged for each Rinne positive and each Rinne negative group, and for the air acuity curves both ears were included.

The averaged bone acuity curves for the six classes of cases are shown in Fig. 1. It is at once apparent that a close correlation exists between the range heard and the sensitivity recorded in the three groups in spite of the fact that each is made up of a Rinne positive and a Rinne negative component. This implies that the acuity for hearing bone sounds is quite independent of the functional condition of the air sound transmission system, and that the transmission of the bone sounds in these cases is directly through the wall of the otic capsule (cranial).

Comparison of the curves also suggests that insofar as the usual fork range for testing bone acuity is concerned (120 to 512 Hertz), only the cases of marked disability (Group C) show a conspicuous average loss in sensitivity. The normal group (R + A) display a slightly lessened average sensitivity over the fork range when compared with the corresponding Rinne negative group (R — A). This, however, may be attributed entirely to the masking effect of slight adventitious noises which affected the former but not the latter class of cases. The tests were made in a quiet room but not in a silence chamber. Similarly, the slight advantage of the R + A group over that of the R — A group in the higher frequency range may be accounted for on the basis of the air sounds developed directly by the bone telephone and also perhaps by head resonance. However, a comparison of these two curves does not suggest that the Rinne negative cases of group A have the prolonged bone transmission which is described as characteristic for this condition.

The rather marked variations in the C group are in part due to the relatively few Rinne negative cases examined, and also to the wider range of sensitivity shown in these extremely deafened individuals.

The evidence presented in these averaged curves indicates that the bone telephone is an excellent method for determining the functional efficiency of the perceptive mechanism throughout the

audible range quite irrespective of whether the case be Rinne positive or Rinne negative. This statement, however, must be qualified to apply only to the Rinne negative type selected where there is no occlusion of the external auditory canal, and where no loading of the drum membrane with secretion or pus is found.

The averaged air acuity curves for these same groups are shown in Fig. 2. Here it may be noted that the curves for the normal (R + A) and the R + B groups have been somewhat arbitrarily completed. This was necessary because both groups heard some of the frequency range under the conditions of the full attenuation as used.

It is obvious that an arrangement into three groups, each containing a Rinne positive and a Rinne negative component, as found in Fig. 1, has been completely changed. Here all of the cases of Rinne positive fall into one general class, and all of the Rinne negatives into another. The air acuity curves for the R + B and for the R + C groups are quite definitely skewed toward the low frequency end when compared with the normal R — A curve. This skewing is also found in the R — B and the R — C curves when compared with the R — A curve and is equivalent to what has already been noted by means of the bone telephone. Inasmuch as this skewing occurs in both sets of curves, and is quite independent of the Rinne positive and Rinne negative condition, it may be interpreted as due to a similar perception disability. It will also be noted that the R—A curve is nothing more than an evenly depressed normal R + A curve. Comparison of the air sensitivity in the R + B group with that of the R — A reveals a somewhat similar acuity above C4, while below this frequency both the R + B and the R + C cases show a distinct advantage. This point is important in evaluating the fork tests and will be considered later.

A correlation between the air and the bone acuity in the Rinne positive and in the Rinne negative cases should therefore throw light on the efficiency of the air sound transmission system. These correlations are effected in Figs. 3 and 4. It must, however, be remembered that direct quantitative comparisons are not permissible, and that gross differences only may be considered.

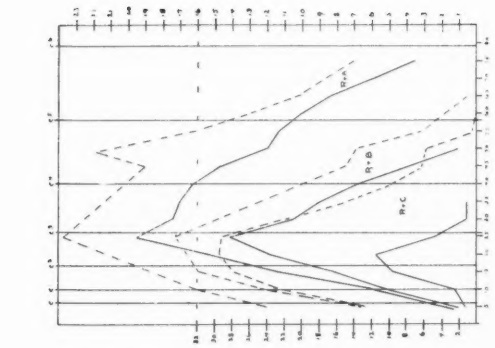

Fig. 3.

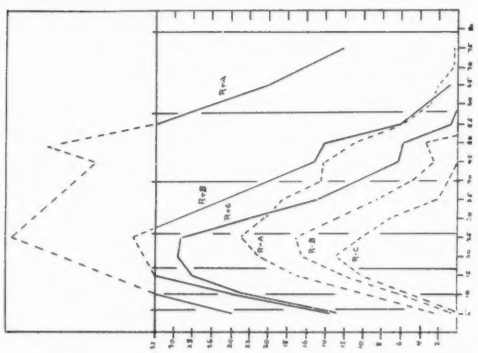

Fig. 2.

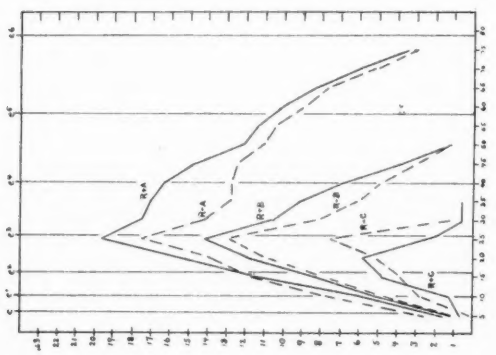

Fig. 1.

Fig. 3 presents the averaged air acuity curves for the Rinne positive groups in broken line plotted against the corresponding bone acuity curves in solid line. It will be noted that the air acuity curve lies at a higher level than that for the corresponding bone acuity, and suggests, if the case reports Rinne positive at any frequency, he may be considered Rinne positive for all frequencies. A report of a change over from the Rinne negative to positive may be due to the differences in the relative air and bone intensities developed by the forks employed, or perhaps also the condition of an almost complete functional loss in the air sound transmission apparatus. It will also be seen that the two curves for the R + B group are closer together than the corresponding curves for the R + C group. This may be taken to indicate that the R + B cases reported for examination because of a beginning presbyacousia with superimposed conduction deafness which exaggerated the deformation of the auditory field sensitivity in the spoken range. The R + C group, on the contrary, show little conduction deafness. The other possible explanations, which suggest themselves, are that senile changes affect the bone acuity more than the air acuity or that the efficiency in the transmission of bone sounds is relatively less pronounced for intense signals than in the case for the air sounds. It is unfortunate that individuals may have a hearing loss of from 10 to 15 sensation units without being definitely conscious of a hearing disability, and it is a tinnitus or a sensation of fullness in the ears which brings them to the otologists for examination.

Fig. 4 shows the correlations between the air acuity (broken line) and the bone acuity (solid line) in the corresponding Rinne negative groups. Here it will be noted that, according to this method, the air acuity curves lie below the corresponding bone acuity curves. The exception in Group C may be attributed to the few cases and to the wide variation in these cases as has been already pointed out. The close correlation suggests that the two tests are similar tests: the one dependent on the sensitivity of the perception mechanism as determined by direct transmission of air sounds through the medial tympanic wall; and the other, that obtained by direct transmission of bone sounds through the wall of the otic capsule. The lowering in the bone sensitivity is,

therefore, accompanied by a similar lowering in the air sensitivity. This implies that, like the Rinne positive cases, if the report is negative at any frequency, it is likely to be negative at all frequencies.

We may be permitted to assume, on the basis of what has been presented, that the Rinne negative cases of Group A have a practically normal perception apparatus, while the sound transmission system is to all intents and purposes quite functionless. We may also assume that in these Rinne negative individuals the air acuity is directly comparable with bone acuity. If, therefore, a Rinne negative of this type, with normal perception apparatus, is tested for air acuity, the amount of increased intensity required over that of a normal individual represents the functional efficiency of the normal air sound transmission, and at the same time indicates the degree of acoustic insulation of the normal labyrinth against the direct transmission of air sounds. The Rinne negative cases of Group A show an average lowered air acuity over the entire range of 35 to 40 sensation units. A line drawn to represent this acuity on the Western Electric chart corresponds fairly well with the bone acuity normal shown on these records. This fact is not without practical significance, because if the air tests on these Rinne negative cases are directly comparable with bone acuity, then the observations made with an air receiver are just as good a criterion of the functional condition of the perceptive apparatus as those made with the bone telephone and with the additional advantage of being able to test each ear more nearly separately.

The almost uniform depression of the air acuity curves from that of the normal in these Rinne negative cases may be construed as direct evidence of the almost equal functional efficiency of the air sound transmission apparatus throughout the audible range. The intensity increase required for a direct transmission of air sounds through the medial tympanic wall over that for activation through the normal sound transmission apparatus indicates that these two routes cannot be simultaneously operative in a physiologic sense. This is true because the normal threshold intensity by direct transmission of 1 is added to that already arriving through the drum membrane and ossicular

chain (3000), and the contribution would not be sufficient to come to consciousness.

Accordingly, if a normal individual had the drum membrane and outer ossicles removed, he would be deafened by a factor of perhaps 30 sensation units. The figure is placed somewhat lower than in the Rinne negative cases because in the former the stapes would be movable.

The statement that the sound transmission apparatus appears to be of about equal efficiency throughout the audible range is seemingly in opposition to the more or less accepted view that conduction deafness affects the low frequency range more seriously than it does the high frequency end of the audible field. Let us assume that a patient of the R — A type is examined by an older individual of the R + B type. The examiner's interpretation on the basis of a comparative test would be that the patient hears the higher frequencies well and the greatest loss is at the low frequency end. In point of fact, the comparison includes the physician's own degree of presbyacousia and the observation may therefore not be considered a real phenomenon. So far as a diagnosis of a conduction deafness is concerned, the result is the same. It has been the writer's own experience that the R — A cases examined all hear the high frequency range better than he does himself and his own acuity curve is quite typically of the R + B type. In spite of this fact, the air acuity is normal for both ears up to at least 2048 Hertz, and he is not conscious of the slightest auditory handicap under usual conditions.

There is, of course, a physical possibility that the efficiency of the air sound transmission apparatus is relatively greater at the lower intensities because the elastic annular ligament at the stapes footplate is relatively narrow and may, therefore, yield more readily to minute movements than to larger excursions. This limitation effect should be less pronounced at the round window.

It must also be remembered that the acuity for hearing bone sounds is more variable in normal individuals than that for air sounds, and this fact must always be borne in mind in the correlations of air and bone acuity for purposes of a differential diagnosis.

The possibility of the separation of the indirect from the direct air transmission has already been considered, and all of the cases thus far reported belong to the type of cranial transmission of bone sounds. There is, however, another route through which bone sounds may activate the cochlea, and perhaps the comparison of two cases may serve to indicate what is meant.

A young man with history of chronic progressive deafness showed no lateralization for either the 256 or 512 forks from the vertex; Rinne negative on the right and Rinne positive on the left. The air and bone acuity curves are presented in Fig. 5. The bone acuity curve (broken line) lies within the variable normal to about C3 with a rapid drop-off in sensitivity to about 50 and resembles that of the B group of Fig. 1. This may be taken as evidence of a perception disability particularly at the high frequency end of the audible range. The air acuity curve for the right ear is shown in solid line and that of the left ear in a —x— line. The left air curve is like the R + B group, while that of the right ear is quite similar to that of the R — B group of Fig. 2. The loss in sensation units on the left was 20 at 256 and 512; and 25 at 1024 Hertz, which is not sufficient to produce a Rinne negative response. The loss of the right was 40, 45 and 50 sensation units at these frequencies, respectively, which is sufficient loss in air acuity to elicit a Rinne negative response with the amount of bone acuity indicated. This case may, therefore, be regarded as one of a beginning perception disability affecting the high frequency end of the audible field, with complete functional loss of the drum membrane and ossicular chain on the right, and with marked conduction deafness on the left. Possibly the condition on the left may go over later into that found on the right. In spite of these differences in the efficiency of the air sound transmission apparatus, no lateralization was noted.

The second case is that of a young woman with a chronic otitis media on the left which had been discharging for many years. She developed an earache on the right and introduced warm vaseline on the advice of a friend; "Went deaf"; and reported for examination. The Rinne was negative right and left with no evidences of a lateralization. The air acuity curves, taken at this time, are shown in Fig. 6 with that of the right ear

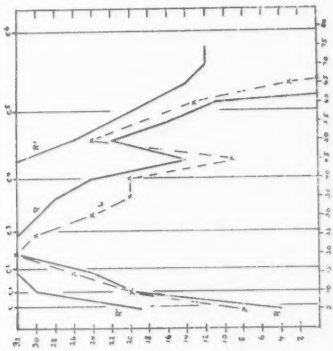

Fig. 6.

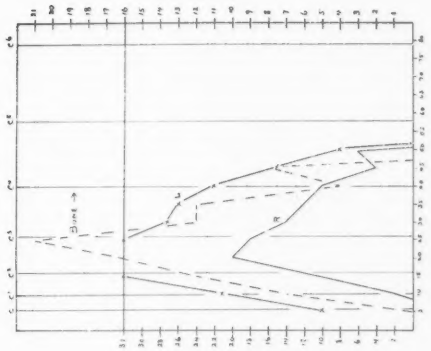

Fig. 5.

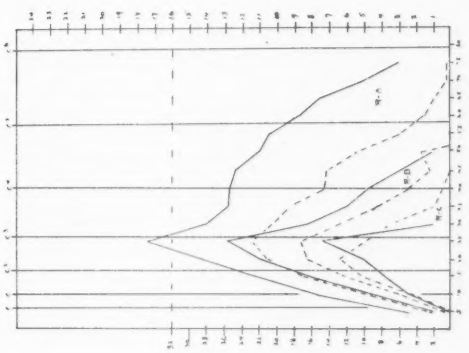

Fig. 4.

as a solid line and that of the left as a —x— line. In a general way, the air acuity is quite similar on the two sides. Removal of the vaseline brought the right ear acuity to practically normal (R1). This case, therefore, seems to indicate that loading of the drum membrane on the lateral surface with vaseline has an effect which is similar to loading the membrane with pus on the deep surface. The more marked loss in air acuity at the high frequency end has been noted by Kranz and myself[2] in the normal cases tested with water in the external auditory canal or under conditions of occlusion.

The bone acuity curve is shown in Fig. 7 as a broken line. Against this are plotted the normal curve taken from Fig. 1, and also the minimum and maximum sensitivities reported in these sixteen cases, quite irrespective of the individuals. It is at once obvious that this case shows a prolonged bone transmission, particularly through the usual fork range. This is also indicated by the fact that the degree of loss in air acuity is not sufficient to produce a Rinne negative response, and therefore the bone acuity must be enhanced to bring about the result. Removal of the vaseline from the right ear increased the air acuity as it decreased the bone acuity because now the subject lateralized distinctly to the affected side.

The comparison of the curves in these two cases indicates that in the first individual the bone acuity was not materially affected by the differences in the air acuity on the two sides. The bone acuity curve may, therefore, be considered dependent on the sensitivity of the perceptive apparatus as determined by direct transmission of the bone sounds through the wall of the otic capsule. The second case, however, shows a definitely prolonged bone transmission which affects the lower frequency range, and which has been brought about by the loading of the drum membrane on the one side with vaseline and on the other with pus. The lowered air acuity on the left in Case 1 (Fig. 5) is practically the same as the loss in both ears of Case 2 (Fig. 6), and yet the former reported Rinne positive and the latter Rinne negative. Accordingly the bone acuity in Case 2 must have been increased, because a 25 sensation unit loss in air acuity with normal bone conduction is not sufficient to produce the Rinne negative re-

sponse. Removal of the vaseline from one ear increased the air acuity to normal and at the same time decreased the bone acuity because now the patient definitely lateralizes to the affected side and is Rinne positive on the right.

Kranz and the writer[3] performed the first quantitative experiments on the correlations between air and bone acuity at Riverbank Laboratories. Briefly, we found, when the external auditory canals were occluded, that the blocking effect on air sound was more pronounced in the upper frequency range. Bone acuity, however, under these same conditions, was positively enhanced through the lower part of the frequency range over that normally recorded in the silence chamber. A somewhat similar, although less marked effect on bone sounds obtained under conditions of partial occlusion, and this information is set forth in Fig. 8. Practically the same result obtained on repeating the water filling experiments of Wheatstone. We also determined that both air and bone acuity are most pronounced at zero pressure in the air of the external auditory canals, and both were unfavorably influenced by increases and decreases in the air pressure. We arrived at the permissible interpretation that the evidence favored the transmission of both air and bone sounds by way of the drum membrane and ossicular chain. We also found, in a few deafened cases examined, who would properly belong in the R — A group described here, that the acuity for hearing bone sounds was not materially affected by occlusion of the external auditory canals.

The material presented in this paper indicates we overlooked one point in studying the effects of variations in air pressure on bone acuity. We sealed a binaural stethoscope into the external auditory canals and this resulted in an enhanced bone acuity which we ourselves had demonstrated comes about through an incomplete and a complete occlusion. It is this prolonged bone transmission which takes the craniotympanic pathway, and it is this prolonged bone transmission which was affected by the pressure changes. Accordingly a craniotympanic transmission for bone sounds does not occur, insofar as the evidence shows, excepting under conditions of partial and complete occlusion of the external auditory canal or where the drum membrane has been

loaded on the lateral or medial surface; and then only in the presence of a functional air sound transmission apparatus.

Under normal conditions the auditory end organ may be regarded as acoustically insulated, and the sound transmission apparatus may be considered a mechanical transformer which matches the impedance differences between air and water. The internal ear, therefore, occupies a position of relative quiet and the air sounds are brought to it by the drum membrane and ossicular chain. However, in the case of bone sounds, the situation is somewhat different, because here the vibrations are already passing through the otic capsule, and the contribution to the intensity through addition by way of the sound transmission apparatus under normal conditions may be insufficient to come to consciousness. The fact that the intensity ratio of 200 to 1 is found on comparing the average sensitivity at 512 Hertz under normal conditions with that necessary under conditions of complete occlusion lends weight to the contention that the two routes for bone transmission are not simultaneously operative in a physiologic sense.

This information may have a practical application, because if a patient reports an air acuity which resembles a typical presbyacousia with greater losses in sensitivity above 2048 Hertz and a decrease of say 20 to 25 sensation units below this frequency and at the same time is Rinne negative, the tentative diagnosis of a normal perception apparatus and an efficient sound transmission system with drum membrane loaded with secretion or pus may be suggested.

That wide differences in opinion exist on the subject of prolonged bone transmission is indicated in an extensive bibliography on the subject, and perhaps these differences may be accessibly presented in the discussion on this topic at the International Congress meeting at Copenhagen in 1928. The discussion was on a paper by Retjo[4] and was quite inadvertently precipitated by my statement on prolonged bone transmission. The following is the discussion in full:

Pohlman. "May I ask the essayist in what manner he conducts the test for prolonged bone transmission. My own experience is that prolonged bone transmission, like paracousis Willisi, is not

a real phenomenon. One individual, alleged to have a prolonged bone transmission, was tested with several forks and a direct comparison made with myself. Under usual conditions of test, he heard the forks from 5 to 20 seconds longer than I did. However, when the tests were repeated in a deadened room, I heard the forks from 5 to 20 seconds longer than he did. Under usual conditions of test, prolonged bone transmission means nothing more than the masking effects of adventitious noises, and indicates that the test is on the air acuity of the physician rather than on the bone acuity of the patient."

Neumann (Vienna). "Either we have heard something which is really new or there is some mistake in this discussion. I cannot believe that a true bone transmission does not exist. I also cannot see why an otosclerotic patient will show any differences in bone transmission depending on the room in which he is tested."

Frey (Vienna). "We cannot accept the fact that the lengthening of bone conduction or paracousis Willisi should be based on subjective errors. The lengthening of bone conduction is a fact which can be demonstrated at any moment in normal and pathologic cases."

Somerville Hastings (London). "Last year I carefully examined some seventy cases of paracousis by an acumeter so constructed that the sound was conveyed to the ear by means of a well-padded telephone. With this telephone could be sent not only a fine test sound but a noise as well. I found that a few patients heard the test sound better when the disturbing noise was present, and that a few also heard the test sound better when the disturbing noise passed into the opposite ear.

"For ten years I have sought in vain for a patient with increased bone conduction when tested with the tuning fork on the mastoid and with the meatus of both patient and examiner blocked with the finger. I have never found a patient who could hear the tuning fork longer than I could."

Mackenzie (Philadelphia). "May I ask Dr. Hastings a question? If there is no increase of bone conduction in so-called obstructive deafness, how do you explain the following: A patient comes with an acute unilateral middle ear catarrh, for instance, of the right ear. How do you explain the lateralization of

the Weber to the diseased side when the fork is placed in the midline and also far to the left of the midline? In some cases the lateralization to the right side is so pronounced that the patient refers the sound to the right ear even when the tip of the fork is placed on the left mastoid."

Quix (Utrecht). "I must answer Dr. Neumann. I do not maintain that a prolonged bone transmission does not exist, but in all cases in which we have established a prolonged bone transmission in an ordinary room this prolongation has disappeared in an acoustic chamber. Our conclusions have been based on insecure foundations and therefore must be revised."

Shambaugh (Chicago). "I have the impression that the question of prolonged bone transmission is the same as that of paracousis Willisi. I have carried out a series of experiments, using an audiometer, which have demonstrated that paracousis is only apparent. New investigations may make us discard formerly accepted conclusions."

Struycken (Breda). "Has never seen a case of physiologically prolonged bone transmisison. One will notice, if tests are made under conditions of absolute quiet, that deafened people show a somewhat decreased bone acuity."

Mackenzie (Philadelphia). "Did I understand you to say, Dr. Pohlman, that increase of bone conduction in middle ear disease exists only in the imagination of the examiner? Yes. Anyone who has visited me knows we are very careful in our technic. In the instance of a case of acute exudative catarrh and the air conduction is reduced and the bone conduction is improved, these findings are checked up by my two associates, Dr. A. V. Mackenzie and Dr. W. G. Shemley, who are both experts. The findings cannot be doubted. Further, if following treatment by Politzer inflation or paracentesis or otherwise, the patient experiences an improvement in hearing to normal, to tell me that the bone conduction findings before the treatment did not show a lengthening over the normal second finding, exists only in my imagination, is absurd."

Somerville Hastings. "In reply to Dr. Mackenzie, if a patient has one ear blocked with wax, he will not hear better by bone conduction compared with the other ear, provided both ears tested

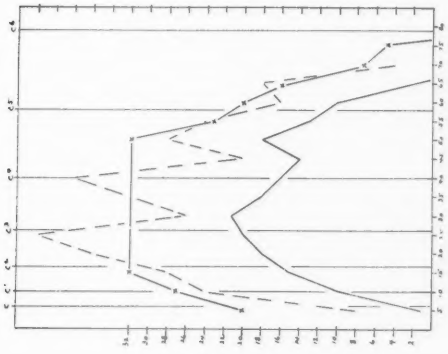

Fig. 9.

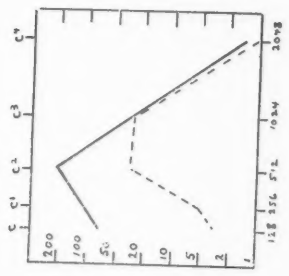

Fig. 8.

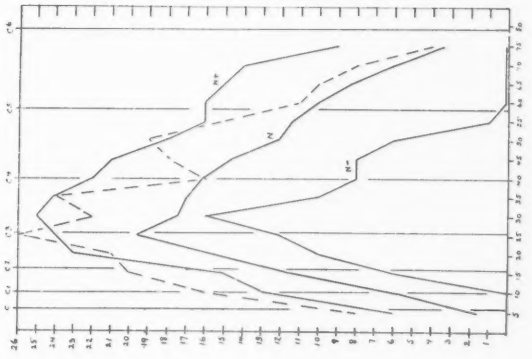

Fig. 7.

of both observer and patient are blocked with the fingers. It is probable that in bone conduction the skull vibrates as a whole and some normal persons hear the tuning fork best on the right side when it is placed on the left side of the skull, but never in my experience when placed on the left mastoid."

Mygind (Copenhagen). "We must not forget the individual variations of bone conduction in normal individuals."

The discussion was closed by the presiding officer and I was not permitted to reply. I am, therefore, taking this occasion to try to clear up the situation.

The discussion reveals that the term "prolonged bone conduction" includes several quite distinct entities: the normal range of variability; the masking effect of adventitious noises; and finally, a comparison of a more efficient craniotympanic bone transmission with a less efficient direct cranial conduction.

Practically all investigators who have employed quantitative methods have mentioned the relatively greater variability in bone acuity over that for air acuity in normal individuals. The normal curve for bone acuity may, therefore, only be established by averaging a series of normal cases. A normal individual with greater than normal sensitivity for bone sounds may have an entirely different interpretation on comparing his own bone acuity with that of the patient than a normal with lower than average sensitivity. The reasons for this greater variability in bone acuity than in air acuity are not entirely explained, but probably the factors of greater variability in the transfer of the sounds from the source; certain anatomic differences in the anatomy of the auditory apparatus, notably in the external auditory canal; and the less acute observation under conditions of the bone test, especially where the sounds are heard by both ears, rather than by one, must be mentioned. In any event, every otologist, making routine comparative fork examinations, should have his own air and bone acuity definitely established.

The second factor mentioned was the masking effect of adventitious noises. It is surprising how even slight and almost unappreciable noises mask the sensitivity for hearing bone transmitted sounds, particularly in the lower frequency range. The English otologists, as indicated by Hastings' statements, have

attempted to eliminate this masking effect of noises by making comparative tests with the external canal of both patient and examiner occluded with the finger tip. Hastings is quite convinced that the entire result is dependent on masking, and that a prolonged bone transmission under these conditions does not obtain. This opinion is shared by quite a few others. The material presented here does not deny the importance of the masking, but it opposes the "absolute bone conduction test" on the basis that it is craniotympanic transmission and is, therefore, prolonged bone transmission. Masking explains the lateralization to the conduction deaf side not because a conduction deafness increases the bone transmission to that side, but because it decreases the acuity of the opposite side. This is in perfect agreement with the view expressed by Shambaugh.

Recently Hallpike[5] published a paper in which he reports the results of fork tests on deafened cases under usual test conditions and also in a silence room. He found "normal hearing individuals who succeed in localizing the tuning fork upon the vertex to the occluded ear in an ordinary room do so with almost equal certainty in absolute silence. If, however, they are kept unaware of the side of the occluded ear by using two similar earpieces, one of which has the lumen closed, then the power of localization becomes extremely uncertain."

Information which bears on this particular point has been already published by Kranz and myself. We found that lateralization to the occluded side occurred in a silence room. We also found that partial occlusion produced an enhancement effect at 512 Hertz, although not so marked as in the case of complete occlusion. Hallpike compared the acuity under conditions of complete occlusion with that with auditory canal normal, as shown in the first statement quoted. The second and conflicting statement, however, may be explained on the basis that he was comparing a partial with a complete occlusion, where the difference is much less. This information is presented in Fig. 8 of this paper. The elimination of the masking, therefore, does not explain all of the facts. If a normal individual or a Rinne positive case is tested for lateralization in a silence room he will respond quite promptly to the occluded side or to the side on

which the external auditory canal has been filled with water, provided the sensitivity of the labyrinth on the two sides is the same. Prolonged bone transmission is a real phenomenon where the sound transmission system is functional under at least these two conditions: occlusion of the external auditory canal and loading of the drum membrane. This conclusion is, therefore, in full accord with that made by Mackenzie for the type of case reported.

In this connection a statement by Runge,[6] who has experimented extensively with bone transmission, is indicated.

"Apart from air transmission, we also hear by direct bone transmission when the air sounds deliver their vibrations directly to the bone, or by indirect bone conduction when the air pulses produce vibrations in the cranial bones which in turn are transmitted by them to the labyrinth. According to Bezold, the transmission of low tones takes place only through the ossicles, while the high tones pass directly through the otic capsule to the basilar membrane. As opposed to this viewpoint, a number of investigators claim the possibility for the transmission of all frequencies by bone conduction directly to the basilar membrane. Some time ago I was able to establish, what had been considered probable by a number of observers, that both routes, through the ossicular chain and also the direct route through the bone to the basilar membrane, might be selected at all frequencies. It is also possible to calculate about how much each of these two routes may contribute to the intensity. Accordingly the ossicular chain is by far the more important route for the transmission of low tones, and is less important for high frequencies because of the much greater sensitivity of the perceptive mechanism at such frequencies."

The foregoing statement appears opposed to what has been submitted in this paper. However, on careful analysis it develops that the differences in opinion are largely dependent on terminology. What Runge calls a direct bone transmission, I have spoken of as a direct air transmission where the air sounds are conducted directly through the medial tympanic wall, probably through the round window. What Runge terms the indirect bone conduction is considered under the name of direct bone trans-

mission, where the vibrations of the cranial bones affect the labyrinth directly through the wall of the otic capsule. Runge finds that all frequencies may pass by either of these two routes, and this was first demonstrated quantitatively by Kranz and myself. However, Runge does not consider a separation of a direct cranial transmission from a craniotympanic pathway. His statement that the drum membrane-ossicular chain route is more efficient in the transmission of low frequencies holds for what is here called a craniotympanic transmission and the result is dependent on his water-filling tests which produce this condition. The statement does not hold for the direct cranial transmission. We differ somewhat on the point of a response in the basilar membrane, because Runge believes that the bone vibrations pass over directly into the membrane, whereas my evidence is that the entire contents of the labyrinth responds to the vibrations passing through it. We also differ on why the low tones in bone sounds pass in by way of the ossicular chain, because Runge believes this may be explained by the greater sensitivity of the internal ear at the higher frequencies, while I hold an abnormal factor, such as occlusion of the canal or loading of the drum membrane, is responsible for this craniotympanic phenomenon. When the external canal is occluded or the drum membrane loaded with water a greater blocking occurs for the transmission of air sounds at the higher frequencies. If this condition resulted from mechanical interference in the passage of sound pulses to the ear, then a similar effect should be noted on the enhanced bone acuity because, according to the "Abfluss" theory of Mach, it is the prevention of the "escape" of air sounds developed in the middle ear under conditions of bone conduction which explains the phenomenon. Accordingly air sounds of low frequency developed by bone transmission should "escape" more readily than those resulting from higher frequencies and the reinforcement to bone acuity should be most marked at the high frequency end. Inasmuch as all reinforcement occurs at the low frequency end, the Mach explanation cannot obtain. There is a suggestion that the effect is dependent on the vibration of the plug or of the water itself, and evidence for this will be submitted at another time. The Rinne negative cases, here classified

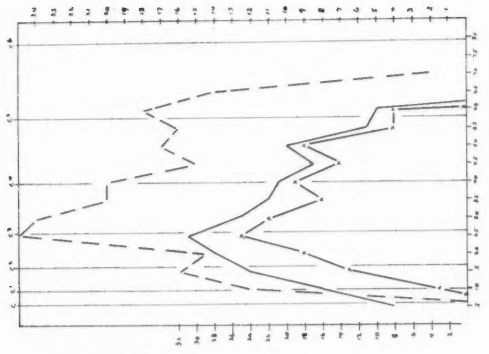

Fig. 12.

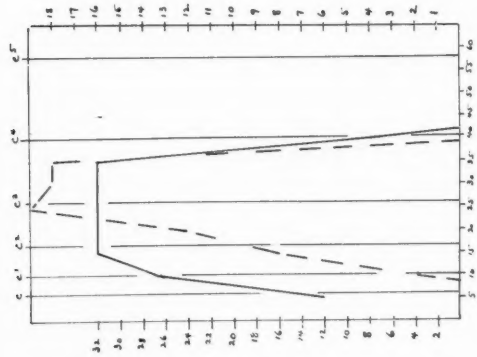

Fig. 11.

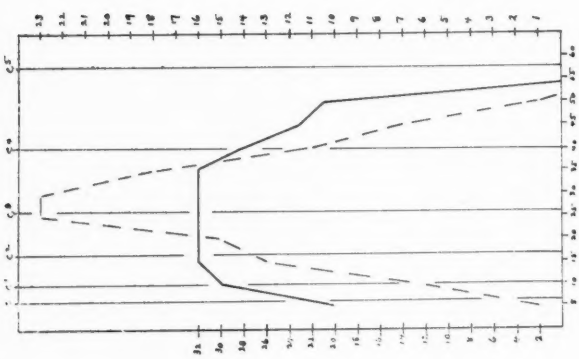

Fig. 10.

as those with a nonfunctional air sound transmission apparatus, do not show the enhanced bone transmission (prolonged bone conduction) when the external auditory canals are occluded. Neither do they display the lowering in bone acuity, in the fork range, characteristic of all Rinne positive cases, under conditions of decreased pressure in the air of the external auditory canals. This implies that the reason for the Gelle and Lucae tests is not dependent on a change in the intralabyrinthine pressure but may be attributed to the same cause as that responsible for the Rinne negative report—a nonfunctional air sound transmission apparatus.

The results presented here are also quite in agreement with the work of Fowler, excepting that Fowler favors the craniotympanic route for transmission of all bone sounds, while I have attempted to separate this route from that of a direct cranial transmission. Fowler is also quite correct that a Rinne test is quite unnecessary where audiometric methods for air and bone sounds are available. However, the test is valuable when such methods are not available.

The material presented in this paper indicates that the direct air transmission acuity is comparable to direct bone acuity and is a fair criterion of the functional efficiency of the perceptive mechanism. The indirect air acuity, however, also includes the factor of the efficiency of the air sound transmission apparatus. A comparison, therefore, of the direct bone acuity with the indirect air acuity should be helpful in the determination of the conduction and the the perception component found in the cases of mixed deafness. The following cases are reported briefly to show the correlations between the air and bone acuity and their diagnostic significance.

Fig. 9 shows a normal bone acuity curve (broken line) with normal air acuity on the left (—x—). The air acuity curve on the right (solid line) indicates a complete functional loss of the air sound transmission apparatus. This case lateralizes to the right, probably because of the masking effect of slight adventitious noises on the normal ear (false lateralization). Occlusion of both external auditory canals changes the lateralization to the left. This may be explained by the elimination of masking noises

and also because of the craniotympanic bone transmission developed by the normal ear under these conditions. The diagnosis is normal perceptive apparatus on both sides with normal sound transmission system on the left and complete functional loss on the right (probably ankylosis of the stapes).

Fig. 10 presents a beginning presbyacousia in the bone acuity curve (broken line) as shown in practically normal responses at the low frequency end with limitation at the high end of the audible scale. The air acuity was practically the same for both ears and only one is shown as a solid line. The low frequency air sensitivity is practically normal, which may be taken as evidence that the case has no conduction deafness. The air acuity curve drops off in sensitivity in a manner like that of the bone acuity curve. The diagnosis is, therefore, a beginning disability of the internal ear with no conduction deafness.

Fig. 11 shows a bone acuity curve (broken line) which is slightly lowered in the low frequency end and which drops off quite abruptly at about C4. The slight loss in air sensitivity (both ears alike and only one shown) at the low frequency end of the range has already been noted in the bone acuity curve and indicates that the sound transmission apparatus is probably about normal. The air acuity curve shows the same drop off in sensitivity as that found in the bone curve. The diagnosis is, therefore, a profound loss in sensitivity in the internal ear above C4.

Fig. 12. Here the bone sensitivity (broken line) is about normal throughout the range tested with complete functional loss in the air sound transmission system on both sides (acuity for left ear —x— and for the right as a solid line). The case may be regarded as one of complete conduction deafness with normal labyrinth on both sides.

Fig. 13 shows an obviously prolonged bone transmission (broken line) with complete conduction deafness on both sides. The drum membranes in this case are very much thickened, and it is the outstanding exception to the rules proposed. It is possible that the case may be like the condition reported by Runge with bony ankylosis of the malleus to the roof of the tympanum.

Fig. 14 presents the unusual conditions of pronounced lowering in the bone sensitivity below C4, which indicates a disability

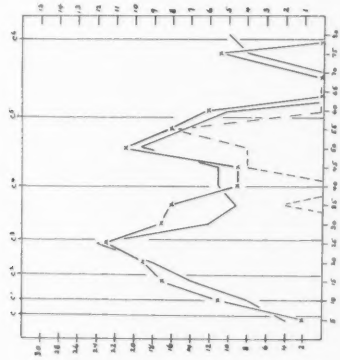

Fig. 15.

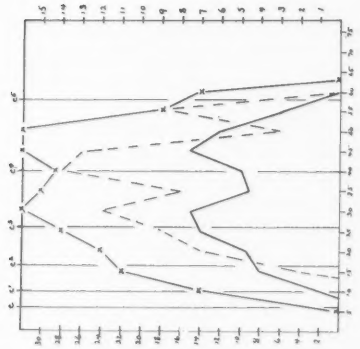

Fig. 14.

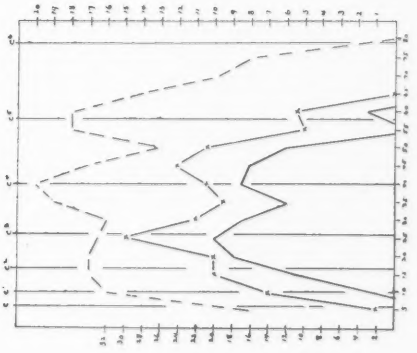

Fig. 13.

in the perception apparatus. The sensitivity curve for air transmission on the left (—x—) may be taken to point to a fairly normal sound transmission system on this side, while on the right (solid line) the evidence is for a complete conduction deafness.

Fig. 15. Here the bone acuity curve (broken line) shows a markedly reduced sensitivity at the low frequency end while the air acuity curves for both ears point to an efficient air sound transmission system.

Fig. 16 contrasts the air acuity curves and the bone acuity curves in the case of a young woman and that of an old lady. The curves on the left are quite similar, with the broken line indicating the air sensitivity of the young woman, the solid line that of the old lady. The bone sensitivity curves on the right, however, show that the hearing disability in the young woman is due to complete loss in function of the sound transmission apparatus, while in the old lady (solid line) the loss in air sensitivity is quite clearly dependent on a lowered efficiency of the perceptive mechanism.

Fig. 17 presents a condition similar to that found in Fig. 15 except that the air acuity loss on the left was too great to be measured by this method. The position of the right air acuity curve (solid line) suggests that the entire disability is located in the internal ear.

Fig. 18 contrasts the air acuity of the better ear in four cases taken with the Western Electric audiometer. The curves have been turned about and the frequencies spaced to conform with the method employed in this paper. The O line indicates the maximum output; the n = O line is the normal air acuity line, and the line marked B represents the acuity for bone sounds as determined by their method. The air acuity of I is practically normal up to C3 with rapid drop-off in sensitivity above this level. This may be taken to indicate that no conduction deafness is present. Cases II, III and IV tend to follow the bone acuity curve B, and so far as the air acuity is concerned seem to be about the same sort of thing.

Fig. 19 shows the bone acuity curves of these cases taken with this apparatus. Case I is clearly an internal ear disability with no conduction deafness. Case III is a profound conduction deaf-

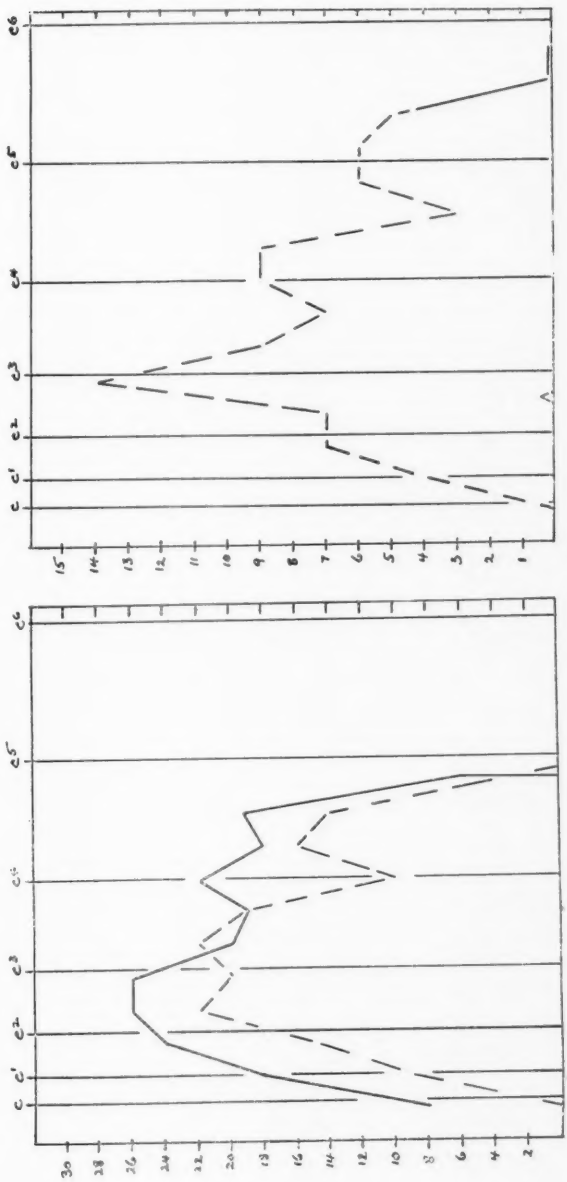
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Fig. 16.

ness with perhaps slight lowering in the sensitivity of the internal ear but not beyond the variable normal. Cases II and IV, however, show little or no conduction deafness and are unusual instances of perception disability most pronounced in the lower frequency range.

These cases answer the question of doubt raised by Kranz and myself[8] on the possibility of a differential diagnosis of conduction from perception deafness in the affirmative. This information, however, cannot be obtained when a comparison is made between the indirect air acuity and the craniotympanic bone acuity, because in both the efficiency of the sound transmission system and the sensitivity of the end organ are involved.

The writer, in closing, wishes to express his appreciation of the courtesy of Dr. J. F. Fairbairn of Buffalo in placing the facilities of his audiometric laboratory at his disposal and making possible this study on private clinical cases.

SUMMARY.

1. The auditory end organ may be regarded as acoustically insulated against air vibrations because it is immersed in the liquid of the internal ear.

2. The middle ear apparatus may be considered to have the function of a mechanical transformer which matches the impedance differences between air and water in the transmission of vibrations.

3. Comparison of the air acuity curves of normal individuals with those obtained from cases in which the air sound transmission system is functionally out of commission and where the perceptive apparatus is normal, indicates the degree of acoustic insulation and at the same time the efficiency of the air sound transmission system.

4. The efficiency of the air sound transmission system is so great, when compared with that of the direct transmission of air sounds through the medial tympanic wall (round window), that the contributions through the latter to the intensity of the signal developed in the internal ear through the former are too small to come to consciousness. The two routes are, therefore, not simultaneously operative in a physiologic sense. This is shown diagrammatically in Fig. 20.

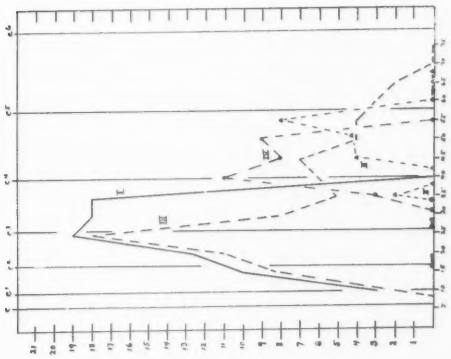

Fig. 19.

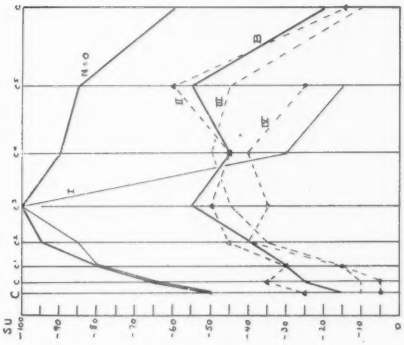

Fig. 18.

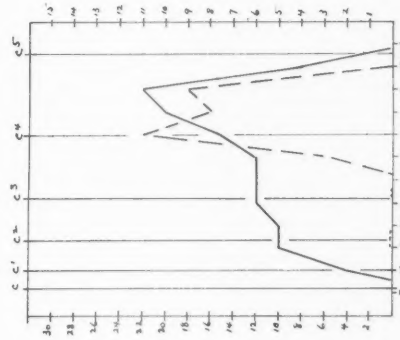

Fig. 17.

5. Where the sound transmission apparatus is at all functional, there the case tends to report Rinne positive, and not until the drum membrane and ossicular chain route is functionally out of commission does a definite Rinne negative condition obtain (Riverbank magnesium alloy fork 256 Hertz). This is true except in cases where the drum membrane is loaded or where the external canal is occluded.

6. The air sound transmission apparatus appears to be of about equal functional efficiency throughout the audible scale, and therefore, if an individual hears any frequency at normal intensity he may be regarded as not having a conduction deafness. The typical curve for conduction deafness is an even depression of the normal acuity curve, and any departures from this must be considered as due to normal variability or to disability in the perception apparatus.

7. Bone sounds are transmitted to the cochlea either through the wall of the otic capsule (cranial) or by way of the drum membrane and ossicular chain (craniotympanic). Under conditions where direct bone transmission is effective, the contributions to the intensity developed in the cochlea through additions by way of the drum membrane and ossicular chain do not appear to be sufficient to influence the threshold stimulus. Accordingly, the bone acuity curve is not influenced by the functional condition of the air sound transmission system, except as noted in 5, and is independent of the Rinne positive and Rinne negative report. See Fig. 21.

8. The craniotympanic route is developed under conditions of occlusion of the auditory canal or loading of the drum membrane in the presence of a functional air sound transmission system. These two conditions lower the air acuity and at the same time enhance the bone acuity (true prolonged bone transmission) throughout the usual fork range. See Figs. 22 and 23.

9. In Rinne negative cases with nonfunctional air sound transmission apparatus, the acuity curve for air sounds is dependent on a direct transmission through the medial tympanic wall (round window), and is directly comparable with the bone acuity curve by direct transmission through the otic capsule. The depression of the air acuity curve is by a factor of 35 to 40 sensation units

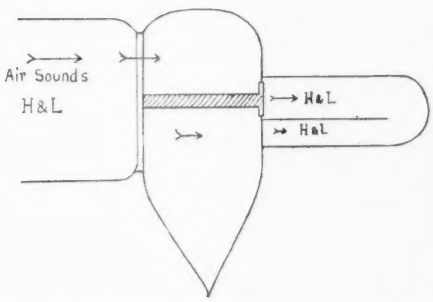

Fig. 20. The length of the arrow indicates schematically the intensity response. High frequencies noted as H; low frequencies as L.

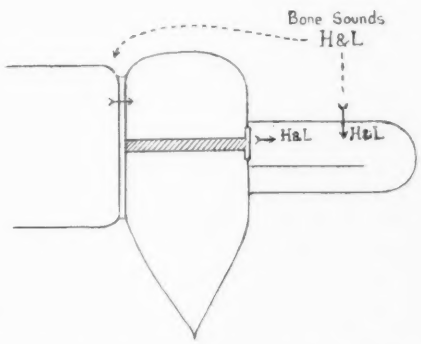

Fig. 21. The length of the arrow indicates schematically the intensity response. High frequencies noted as H; low frequencies as L.

throughout the audible range if the perception apparatus is normal. The tests for air sounds by forks or by telephone are similar to bone tests in these cases and furnish information on the condition of the perception apparatus in either ear.

10. The "absolute bone conduction test" is performed under conditions of occlusion of the external auditory canal and presents a craniotympanic type of transmission which is equivalent to a prolonged bone transmission. This occlusion, like the condition where the drum membrane is loaded, deforms the normal acuity curve for both air and bone sounds, particularly through the fork range.

11. The correlations between the air and bone acuity curves in Rinne positive cases makes it possible to evaluate, in part, the conduction and the perception component in mixed deafness.

12. The Rinne test should be of great value in the differential diagnosis, provided a standard fork is employed under proper conditions, because where the case reports Rinne positive at one frequency, he may be considered as Rinne positive for all frequencies, and this also obtains for the Rinne negative response.

13. The acuity for hearing bone sounds is far more variable than that for hearing air sounds, even under conditions of a test chamber. This must always be borne in mind in the differential diagnosis. The masking effect on bone sounds, particularly those of low frequencies, of even slight adventitious noises, cannot be overemphasized and for accurate tests a silence room is quite essential for all Rinne positive cases.

14. Lateralization of bone sounds occurs:

(a) To the side of greater sensitivity in the perceptive apparatus (direct cranial transmission).

(b) To the side where the drum membrane is loaded or the external auditory canal is occluded (craniotympanic or prolonged bone transmission).

(c) To the side of conduction deafness, where the effect is due to masking of the better ear (false lateralization).

15. The correlations between the air and bone acuity curves obtained by audiometric methods are quite in agreement with

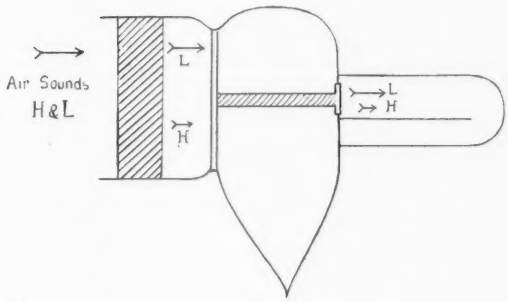

Fig. 22. The length of the arrow indicates schematically the intensity response. High frequencies noted as H; low frequencies as L.

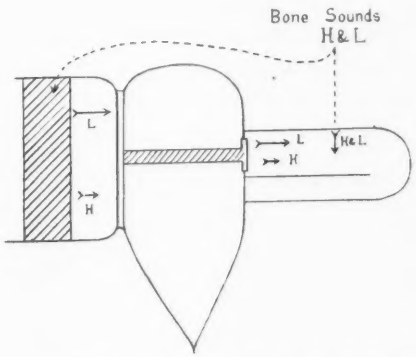

Fig. 23. The length of the arrow indicates schematically the intensity response. High frequencies noted as H; low frequencies as L.

those obtained through fork tests except in conduction deafness. Here the differences are explained by variations in the intensity of air and bone sound delivered by various forks; by the greater minimum-maximum sensitivity of the internal ear in the upper fork range; by the masking effect of office noises; and by the degree of presbyacousia in the examiner.

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LXVI.

A PROVEN CASE OF BRAIN ABSCESS COMPLICATING MIDDLE EAR SUPPURATION: METHODS OF DIAGNOSIS.

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PHILADELPHIA.

This patient consulted us in the outpatient dispensary service complaining of vertigo. Two years prior to the examination he had a violent attack of vertigo which incapacitated him for a few hours. These attacks recurred once every two or three weeks. His last attack of vertigo occurred two weeks prior to the examination. The right ear had been discharging for many years, but he had no other complaints, no headaches, no diplopia, no weakness, and was able to follow his occupation—that of a waiter. The right tympanic membrane presented a large central perforation, with a moderate amount of mucopurulent discharge. There was very little odor to the secretion. No polypi present. No ossicles visible. No evidence of cholesteatoma. The examination of the nose and throat was negative. Hearing in the right or suppurating ear was practically gone, while the hearing in the left ear was normal. There was nothing in the ordinary clinical examination of this patient to indicate that one was dealing with anything unusual, or that the case required more than local cleansing treatment. A vestibular examination, however, elicited findings which were decidedly abnormal—the abnormalities being of the type observed in cases with intracranial lesion. The vestibular findings may be summed up as follows:

There was no spontaneous nystagmus in any direction, no spontaneous past pointing or falling. Pelvic girdle movements good. The nystagmus on turning to the right was horizontal and of 24 seconds' duration. The nystagmus on turning to the left was horizontal and of 12 seconds' duration, which suggested a nonfunctioning right horizontal canal. The vertigo after turning was diminished, being of 19 seconds' duration after turning to the right and of 13 seconds' duration after turning to the left.

The past pointing was especially poor—the left arm persistently pointing in the wrong direction. The caloric test on the right side failed to elicit any responses from the vertical or horizontal canals, showing that the entire right ear was practically nonfunctioning. The left ear—that is, the good ear—when douched showed no responses whatsoever from the vertical semicircular canals, but a good active response from the horizontal canal. On the strength of these findings the patient was advised to enter the hospital, but refused, insisting he was not sick, and would like to continue with his work. The most we could induce the patient to do at the time was to consent to undergo the various consulting examinations. Thus an eyeground examination was made and was entirely negative, and the fields of vision were normal. The Wassermann test came back faintly positive. X-ray of the mastoids showed a sclerotic right mastoid. The urine was normal and the general medical examination showed no pathology of any moment in the lungs, heart or vascular systems. Another vestibular examination was made a month later and findings were substantially the same, with the exception that vertigo after turning was considerably diminished, and the right ear (previously totally nonfunctioning) did show some trace of response. Patient was again advised to enter the hospital and again refused, but was induced to have a neurologic examination with the following findings:

"No hypermetry in finger to nose test. Moves both sides of face equally well, except perhaps muscles around right side of mouth. Tongue protruded straight in median line. Pupils equal and respond to light and to accommodation. No involvement of sensory portion of the fifth. No masseter palsies. Tongue very much coated. No adiadokokinesis in legs or arms. No Babinski. No ankle clonus. No sensory change of any kind. No Koenig's. Gait not indicative of any cerebellar disease. Knee jerks sluggish, no stiffness of neck. In other words, beyond the somewhat sluggish reflexes the entire neurologic examination is practically negative."

From the otologic standpoint we were confronted with a case of chronic ear suppuration on the right side, with vertigo. This vertigo could have been the result of either a direct invasion of

the labyrinth by the middle ear suppuration, or of a toxic irritation of the vestibular mechanism, produced either by the suppurating ear or by any other focal infection. There was one finding, however, which could not be possibly reconciled on the basis of either one of the two above mentioned hypotheses—namely, the total loss of function from the vertical semicircular canals in the left ear—the ear which showed no evidence of inflammation, and where the hearing was normal and the horizontal canal responded well. This blotting out of function in the vertical semicircular canals in an apparently normal ear could be produced only by some intracranial lesion. The shortening of the vertigo after turning as compared to the nystagmus was also indicative of some central disturbance. We were confronted then with a case of ear suppuration showing intracranial phenomena. Since these intracranial signs were so definite we were forced to the conclusion that the vertigo was probably produced by some intracranial complication, and insisted therefore strongly upon the patient's admission to the hospital for possible operation. The problem of more accurate localization was rather difficult. There was no doubt that the patient had an intracranial lesion. There was also no doubt that the intracranial lesion was in the posterior fossa, because of the unusually poor vertigo and past pointing. The presence of a suppurating ear, while not conclusive in itself, was strongly suggestive of the fact that whatever lesion was present was in some way related to the suppurating ear. The possible lesions to consider now were these:

Localized meningitis
Purulent labyrinthitis
Cerebellar abscess

Temporal lobe abscess could be excluded, since everything pointed to its being a posterior fossa lesion. One of the outstanding features of the case was a total loss of function in the suppurating ear. Purulent destructive labyrinthitis could easily account for that, but could not account for the signs of intracranial lesion. To be sure, a diagnosis of a double lesion—that is, purulent labyrinthitis, complicated by cerebellar abscess, was not impossible, but one is naturally reluctant in diagnosing multiple lesions when it is possible to explain all of the phenomena on the

basis of one lesion. The total loss of ear function on the right side did not necessarily indicate a lesion of the labyrinth itself, since a lesion involving the eighth nerve could produce the same phenomena or rather lack of phenomena. A collection of pus in the cerebellopontine angle could readily affect the eighth nerve, and being within the cranium could easily account for the intracranial findings on examination. The latter hypothesis seemed especially likely, since the findings on the two vestibular examinations varied somewhat. It will be recalled that during the first examination there were no responses whatsoever from the right ear, whereas on the second examination, which was carried out a month later, the labyrinth did show some slight responses. It was reasoned, therefore, that if this lack of responses were due to a purulent labyrinthitis the destruction would be complete and permanent, whereas, if the eighth nerve was involved in some purulent mass, a slight and temporary recovery could easily occur. For this reason the diagnosis made was a collection of pus in the right cerebellopontine angle. However, the patient refused to come into the hospital. Three weeks later he was brought in semiconscious and died the same night. An autopsy revealed a small cerebellar abscess deeply placed in the right cerebellar hemisphere with a purulent labyrinthitis.

Although the vestibular examination did not localize the lesion accurately in this particular case, it was, however, the only examination which indicated the presence of an intracranial lesion and even pointed to the area affected. It also indicated the need in this case for urgent studies and possibly life saving surgical intervention. This case, in addition to illustrating the inestimable value of a complete vestibular examination in cases with chronic ear suppuration, is also very instructive in aiding in the interpretation of the vestibular findings in such cases.

Thus, the desirability of avoiding diagnosis of multiple lesions, which is true in a general way, does not apply with the same force in cases where the suspected lesion might be a purulent collection. Furthermore, when the cerebellopontine angle region is under discussion as a possible site for an intracranial extension from a suppurating ear, it is probably not wise to assume that the loss of function noted in the ear considered is due to an in-

volvement of the eighth nerve fibers by a pocket of pus in the angle. In other words, whereas, the phenomenon-complex of a totally nonfunctioning ear in a case of known intracranial lesion almost invariably indicates a cerebellopontine angle lesion if the case shows no signs of suppuration or inflammation in the ear, it probably does not hold true in cases where ear suppuration is present. There, it is better to assume that the above mentioned phenomenon-complex is probably produced by a double lesion, namely, purulent labyrinthitis and a brain abscess.

LXVII.

CHRONIC INFECTIONS OF THE NASAL ACCESSORY SINUSES.

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Infection of the sinuses is now the popular fad with the laity. A woman who has an occupational neurosis with a slight mucus nasal discharge now insists she has grave infection in her sinuses and will have all the headaches and pain she can imagine to complete the picture of distress.

At the other extreme is the phlegmatic individual with obstructed nasal breathing, profuse discharge, and probably not complaining of headaches, who wonders why he always feels tired and has no appetite, or may wonder why he has a touch of rheumatism, but does not think that some nasal condition may be the cause of his trouble.

The sinus involvement may run from what could be termed the latent type to the most severe infection with fatal complications.

The theory that chronic infection of the sinuses is primarily due to bacteria, poor drainage and aeration should be modified. Bacterial infection is always the final process.

Primarily the mucous membrane is deranged by one of three factors, namely: 1. Deficiency in vitamins. 2. Allergia. 3. Endocrine imbalance.

Avitaminosis produces changes in the sinuses which give a favorable soil for bacterial invasion. This has been conclusively proven by deficient diet in rats.

Allergia causes an edematous membrane which may later become infected and progress to inflammatory polypoid degeneration.

Endocrine imbalance causes the mucosa to lose its tone and allows invasion of bacteria, and when the infection has become well established correction of the underlying cause will not, as a

rule, cure the condition. Furthermore, the underlying cause cannot always be definitely determined and corrected, because avitaminoses, allergias and endocrines are not yet fully understood.

The cause of chronic infection of the sinuses, therefore, is, first, changes in the mucous membrane produced by avitaminoses, allergias or endocrines, followed by invasion of the bacteria normally present in the nose. To obtain complete relief of the sinusitis means not only surgery where indicated but finding the underlying cause.

In children, if the sinusitis has not advanced too far, a balanced diet will effect a cure.

The allergias are very baffling, because there are so many factors to consider: emanations, such as street dust and house dust, dander, hair, feathers, etc.; foods, intestinal byproducts, and physical agents, such as cold, heat and light.

The endocrine balance is lacking in many of the patients.

In the majority of patients the condition is more severe in the winter, due in a large degree to artificial heat. If the efficiency of workers is greatest between the temperatures of 67 and 72, and rapidly decreases below, and especially above this temperature, it is reasonable to suppose that they are also more prone to sinusitis as well.

Those who work indoors in crowded rooms, where the air is bad and the temperature either too low or too high, especially those with sedentary occupations, are much more prone to sinusitis than those who spend their time out of doors. Exceptions, in this case, are those whose trouble is primarily caused by allergia.

The interior of the normal sinus is sterile, but when the sinus becomes infected the discharge may be detected only by douching. It may be serous, mucus, mucopus, creamy pus or flocculent pus. Sometimes it is so scant that only by centrifuging the return flow and examining it under a microscope will the pus cells be revealed.

The local symptoms are purulent discharge, either anterior or posterior. The anterior discharge suggests the frontals, ethmoids or antra; the posterior, the ethmoids, antra or sphenoids. The character of the discharge should be considered. For in-

stance, that resembling sour milk and which mixes with water is more serious than mucopus that does not dissolve in water. The former, if it persists after repeated washings, especially if there is an odor, indicates serious damage to mucosa or bone.

Nasal obstruction may be observed due to swollen turbinates, thick secretion or polyps. Incidentally a sinus filled with polyps will probably never clear up until they are removed.

Headache, pain and tenderness are not usually present, but when present should be considered serious symptoms. On the contrary, during acute exacerbations they are frequently present.

The area of pain is not a reliable index to the sinus involved. In a frontal sinus the pain is usually about the frontal, radiating over the forehead. If the antra, the pain is in the cheek, teeth, hard palate or temple. If the ethmoids, at the root of the nose and between the eyes, and the vertex. In the sphenoids the pain is about the eyes, vertex, mastoid and occipital regions.

There is rarely swelling of the forehead or cheek. Giddiness and tinnitus may be associated with chronic sinus infection. Anosmia and cacosmia may be a symptom, especially in the presence of polyps or much pus.

Involvement of the ethmoids, I do not believe, can ever be present without change in the mucosa of the middle turbinate. The middle turbinate is a part of the ethmoid bone, and whenever there are changes in the labyrinth they will be noted in the membrane of the middle turbinate. In a normal middle turbinate the mucosa is smooth, thin, pinkish in color and glistens from the normal secretions.

When ethmoiditis is present the middle turbinate changes in color; it is paler and thickened. This thickening resembles a low grade edema along the anterior and inferior border. The turbinate also loses its normal sheen. Later the mucosa of the middle turbinate shows usually a polypoid degeneration, and the typical textbook discharge of pus is more often absent than present, as far as can be determined. The textbooks also regularly divide the ethmoids anatomically into anterior and posterior groups, while practically the factor that produces the ethmoiditis has no regard for anatomy. All the ethmoid labyrinth is involved, with rare exceptions.

The symptoms run the gamut from nothing the patient can observe, and where only the most careful examination will reveal the pathology, to all the symptoms, pain, discharge, fever, local and remote complications.

When the ethmoiditis becomes well advanced there is no trouble in making a diagnosis. There is present, in the hyperplastic type, polypoid degeneration of the middle turbinate, polyps under the middle turbinate, and they may become large and hang down into the nasal space, even to the floor of the nose.

The character and amount of secretion varies from scanty thick mucoid to thin pus. As a rule, when the discharge is thin pus, the mucosa is less thickened and may even show a false atrophy.

A similar pathology may extend to the frontal, antrum and sphenoid sinuses. If there is a profuse flow of secretion it probably indicates antrum or frontal invasion. Here again a like pathology may be present, with only slight secretion, yet the condition causes more harm than when the flow is profuse.

The frontal, when involved, is usually associated with the ethmoids. There is usually only a sensation of fullness above the eye, pain is only present when the orifice is constricted and dams back the secretion, thus causing sudden increase in pressure.

The antrum is more often associated with ethmoiditis than the frontal. The same facts hold true for the secretion, except that the frontal discharge when present in quantity, usually shows in the anterior portion of the middle meatus, whereas with the antrum it may run from the middle meatus over the inferior turbinate half way back or flow backward and first appear posteriorly over the inferior turbinate.

Transillumination of the frontal and maxillary sinuses should be done on every patient with nasal trouble, which, although only an aid, frequently detects trouble in the frontals or antra not otherwise suspected. Some of the conditions which detract from its reliability are: absence of a frontal sinus, and this test, showing dark, would mislead one to suspect trouble, or extremely thick bony walls, which would reduce the glow. In the case of the antra the test may be misleading in the presence of very thick bone, and in children where the tooth buds of the permanent teeth

are under part of the antrum. The test is also doubtful in cases where there has been an old process in frontal or antrum which has healed. Postoperative thickening will also show dark where there is no longer any active process.

In maxillary sinusitis, especially when no other sinuses are involved, the relation of dead, infected teeth to the antral floor should be determined by dental X-rays, and the viability of the teeth tested. Dental infection of the antrum is more prone to have an offensive odor.

Lavage for diagnosis and treatment can rarely be done on the frontal sinus without previously clearing the way through the middle turbinate and anterior ethmoids. The antrum can be washed out via the natural orifice in 70 per cent of the cases, and failing this, by needle puncture under the inferior turbinate. The results of lavage give valuable information as to the quantity and character of the secretion, which, however, may be so scanty that it will be impossible to detect unless caught in a black basin, and sometimes even needs to be centrifuged and studied under the microscope.

The sphenoid may be involved separately, but usually it is part of the ethmoid involvement. Sometimes a string of discharge can be detected running down on the membrane of the ethmosphenoid fissure into the nasopharynx.

When the nasal septum is fairly straight, it is possible to introduce a cannula into the sphenoid sinus and by douching recover the secretion, but in the hyperplastic type there may be little or none.

X-ray films, well made, with special emphasis on "well made," because poor pictures not only are valueless but even misleading, will show the amount and character of the sinus involvement. It is surprising how often involvement of the sinuses is detected by means of the X-ray when it was not suspected from the history or clinical examination.

Prognosis in Chronic Sinusitis.—Generally speaking, patients with chronic sinusitis never recover without treatment. Many have a low grade involvement in which surgery is certainly not advisable, although unfortunately these patients often have been the victims of ill advised surgery. A clear understanding of the

underlying cause would save many patients from this unnecessary surgical meddling.

Sequences and Complications.—The eye and orbit, orbital cellulitis and abscess, retinal, choroid, conjunctiva and iris inflammations, retrobulbar neuritis. In acute exacerbations the middle ear and mastoid may become infected; chronic bronchitis is also a complication. Gastrointestinal disturbances, recurring low grade pyrexia, loss of bodily and mental vigor, psychic disturbances from loss of memory to neurasthenia. Recurring erysipelas, meningitis, frontal lobe abscess. Albuminuria, arthritis, certain cardiac lesions. In general, the sinuses are an important source of focal infection.

Treatment.—In children, removal of the adenoids and tonsils may eliminate the source of the sinus infection. Correction of avitaminosis may place the child in a condition to throw off the infection. The same holds true for the allergic child.

In adults, the vitamins and allergias play some part in prevention, but when the superimposed infection is well established, local treatment, conservative and surgical, is indicated.

Ethmoids that are only mildly involved, slight hyperplasia and secretion with rare exacerbations, should be treated by local applications and the cause removed if possible. This course is also indicated when the frontal sinuses are involved.

In antrum infections, the antrum should be douched with normal saline daily, as long as an improvement in the character and quantity of the secretion is noted. If there is no improvement in the discharge, operation is indicated. All infected teeth in relation to the antrum should be removed, and the infected bone about their roots should be curetted away, taking care not to enter the antrum. If unavoidable, the opening should be as small as possible, and the diseased membrane not disturbed any more than is absolutely necessary, as it will recover after the dental infection has been removed.

The sphenoid should be similarly douched as long as there is progressive improvement.

In well marked, advanced involvement, the above procedure is a waste of time, even in clear cut allergic cases where a complete cure cannot be expected.

Obliteration of the sinuses would completely cure the condition, if such could be done, but unfortunately, only in the frontal sinuses is this possible, and then only with some disfigurement. In sinuses we are dealing with fixed bony walls, with the disease in the lining membrane and rarely in the bone.

Ethmoids that are hyperplastic, causing nasal obstruction and secretion, with a dull, full feeling between the eyes, lack of concentration, with possible extension to the orbit, eye and bronchi, should be removed. If the X-rays show the anatomy of the ethmoid labyrinth to be fairly compact, with not too much extension of the cells over and below the orbit, the cells can be removed completely or nearly so, as well as the whole of the middle turbinate, intranasally.

No otologist would think of making a small opening through the mastoid cortex and blindly curetting the cells. He knows that the chances for a cure would be at a minimum. The same pertains to the ethmoid cells. They are quite analogous, except that the ethmoids were lined with ciliated epithelium before the infection, which subsequently destroyed it. Therefore removal of part of the middle turbinate and curettage of the ethmoid cells will not improve matters but probably make them worse.

If the frontal is also involved, removal of the anterior ethmoid cells will take away part of the frontal floor and increase space for natural drainage of this sinus.

If the sphenoid is involved, the inner wall of a posterior ethmoid cell is usually a party wall with the sphenoid externally. Removing this plus taking down the anterior wall as far as possible, will drain the sphenoid. If there is much hyperplastic tissue, this should be removed, as far as can be reached, with dull forceps.

The antrum, if involved with the ethmoids, or if a separate entity, should be opened by infracting the inferior turbinates and removing the nasoantral wall under this turbinate, as far as the limits of the antrum. If the membrane is polypoid, as much as possible should be removed with angular forceps, and then the anterior and inferior borders of the inferior turbinate trimmed off sufficiently to give free air space to the antrum, when the

inferior turbinate is restored to its normal position. Ninety per cent of the cases can be done intranasally.

The external operation is required in cysts, new growths, fistulæ, extrasinus abscesses, cerebral or orbital complications, and where conservative treatment has failed. This operation is done with a view to removing every particle of infected lining membrane, and as large an opening made into the sphenoid as possible, with removal of as much of the membrane as can be safely reached. The external operation on the antrum, through the canine fossa, gives a complete view for removing the diseased membrane and making a large opening into the nose under the inferior turbinate.

Results.—Much of the discredit placed on the results of sinus operations is due to lack of understanding of the underlying causes. Allergic patients will never be cured, and very little relieved by the most extensive operation, if the sensitizing agent is not found and removed.

From a total of two hundred cases, two-thirds were cured and one-third improved. Cured in this connection means no crusts, pus or polypoid tissue. Improved means relief of symptoms, but some polypoid tissue remains which may be on the turbinates or septum, or on the remaining sinus mucous membrane. Some pus at times and crusts.

39 East Fiftieth Street.

LXVIII.

HEREDITARY DEAFNESS DUE TO BILATERAL ACOUSTIC TUMORS.

A Clinical Study and Field Survey of a Family of Five Generations With a History of Bilateral Deafness in Thirty-eight Members.*

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and

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That hereditary deafness may be the result of bilateral acoustic tumors has not hitherto been recorded. The investigation described in this report was suggested by a statement in the history of such a case which intimated deafness in a number of the family connections for five generations. The survey of this family disclosed a history of bilateral deafness in thirty-eight members, with subsequent blindness in fifteen instances. Of the deaf and blind persons, four were examined prior to death and were found to have choking of the optic discs with secondary atrophy. The seven affected members living were personally examined and presented the clinical findings of bilateral acoustic tumors. The presence of these lesions was proved by necropsy in two cases. Following is the case record of the patient whose history stimulated the investigation:

V A 6, a man, aged 28 (see Fig. 7), was admitted to the neurosurgical service of the University Hospital on October 9, 1928. He was referred to the clinic by Dr. T. K. Wood, of

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Muncy, Pa., who apparently was the first physician to recognize the unique hereditary tendency of the disease in this family. The chief complaint was deafness and blindness.

The history of this patient's illness was essentially a repetition of a common complaint that had affected many members of his family for five generations. The patient stated that partial deafness had begun at the age of 17. Whether or not the onset and progress of the condition in both ears was concurrent is not known. The deafness, however, increased steadily and became complete six weeks prior to admission, coincident with a beginning failure of vision. This failure of vision progressed rapidly, and total blindness was present for two weeks before admission. After the development of complete deafness there had been considerable vomiting. Vertigo and a staggering gait had been present for four months. Whether or not tinnitus was a symptom could not be determined.

On physical examination the patient was found to be a well developed man who showed some evidence of loss of weight. He was totally blind and deaf, but able to talk. He was very drowsy, but when awakened was fairly alert. He was well oriented and co-operative. In order to communicate with him it was necessary to spell out the words on his right palm, using his left index finger to draw the letters.*

On neurologic examination, it was found that the patient's gait was slow and unsteady. He staggered to the right and left, and after walking a short distance he usually fell backward. In the Romberg test, also, he fell backward. Dysmetria was apparent in the finger-to-nose test on both sides, but the heel-to-knee test was well performed. In testing for dysdiadokokinesia, the movements were slow but well co-ordinated. There was no spontaneous nystagmus. The pupils were 4 mm. in diameter and fixed to light. Ocular rotation was limited in all directions, but

*It is of interest that all the members of this family who became deaf and blind used this method of receiving communications from the outside world, provided their education was sufficient to enable them to read and write prior to the onset of blindness. This method of communication has been given the name of "graphesthesia" by Spiller in his lectures to the students at the University of Pennsylvania.

particularly external rotation of the left eye. There was bilateral loss of the sense of smell and total blindness in both eyes. Examination of the fundi revealed a choking of nine diopters in the left eye and eight diopters in the right, with numerous hemorrhages. The corneal reflexes were slightly sluggish. No evidence of involvement of the seventh nerve was found. Both ears were totally deaf. The ninth, tenth, eleventh and twelfth nerves appeared to function normally. The speech was slow, but the words were clearly spoken. All the tendon reflexes were equally diminished, but there were no pathologic reflexes. An audiogram disclosed a hearing loss of 100 per cent in each ear. The blood and spinal fluid Wassermann reactions were negative. Roentgenograms of the skull revealed no abnormalities except a suggestion of erosion of the inner portion of each petrous ridge. Bárány examination by Dr. Lewis Fisher proved the eighth nerves to be entirely nonfunctioning in both the cochlear and vestibular portions.

A diagnosis of bilateral acoustic tumors was made, and suboccipital craniectomy was performed on October 30, 1928, by Dr. Frazier. A large right acoustic tumor was found and partially removed by the intracapsular method. No tumor was seen in the left recess, but a careful search was not made. The postoperative course was stormy, and the patient died three days after the operation from a hemorrhage into the ventricles of undetermined origin.

Necropsy disclosed bilateral acoustic tumors, the larger one being on the right side (see Fig. 1). There were no other tumors on the brain or meninges.

Histologic examination of the tumor tissue by the hematoxylin-eosin stain disclosed interlacing streams of connective tissue fibers with interspersed areas of a looser reticular nature (tissue types *B* and *A* of Antoni). In some of the fibrous streams there was a faint suggestion of nuclear palisading (see Figs. 2 and 3.) On staining by the Gross-Bielschowsky method it was evident that nerve fibers actually had penetrated the tumor tissue and not the capsule alone (see Figs. 4 and 5). The lesions, therefore, were not of the nature of the ordinary isolated acoustic tumor, or perineural fibroblastoma, in which the nerve fibers are present

only in the capsule, but they are of the nature of the true neurofibroma of von Recklinghausen's disease,[2] in which the nerve fibers are found within the tumor tissue proper. The histologic diagnosis was "bilateral acoustic neurofibromas."

In this family the condition has been transmitted as a true mendelian dominant character, as may be seen readily by the charts presented in Figs. 6, 7 and 8. The information regarding the first two generations is rather scanty, and was furnished by the only living member of the third generation (VIII, Fig. 7). It is, therefore, open to question. The information regarding the last three generations, however, was either verified from several sources or by personal examination. To date it has been possible to trace the issue of only one of the twelve members of the second generation.

A most interesting observation in this family is that four children born of affected parents were found to have absent vestibular responses in the Bárány tests with little or no impairment of hearing. This was interpreted as meaning that these subjects had the condition in an early or latent stage of development. It would seem, therefore, to yield clinical corroboration to Henschen's[3] postmortem observation that acoustic tumors originate on the vestibular portion of the eighth nerve.

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Summary of the Observations in Sixteen of the Affected Persons.

Case	Age, Years	Duration Tinnitus: Right, Years	Duration Tinnitus: Left, Years	Duration Partial Deafness: Right, Years	Duration Partial Deafness: Left, Years	Duration Complete Deafness: Right, Years	Duration Complete Deafness: Left, Years	Duration Blindness, Years	Hearing Tuning Fork Tests, Inches: Right 64	Right 256	Right 2000	Left 64	Left 256	Left 2000	Vestibular Reaction, Absent (0), Impaired (1): Horizontal Right	Horizontal Left	Vertical Right	Vertical Left	Method Turning (T) Douching (D) Both (B)	Corneal Reflex Absent (0) Impaired (1) Normal (2): Right	Left
IVE	41	?	12	?	6	23	0	0	0	0	0	0	1	0	0	0	0	0	B	1	1
IVG	61	20	20	?	?	20	20	0	0	0	0	0	0	0	0	0	0	0	B	2	1
IVG2	34	7½	8	7	7½	?	3	?	0	0	0	0	0	0	....	....	....	....	....	....	....
VA3	34	?	?	?	?	13	13	6	0	0	0	0	0	0	0	1	0	0	D	0	0
VA5	30	15	15	?	14	16	0	0	0	0	0	0	2	0	0	1	0	0	B	1	0
VA6	28	?	?	11	11	6†	6†	2†	0	0	0	0	0	0	0	0	0	0	D	1	1
VA7	26	?	?	?	?	3	3	9‡	....	....	....	....	....	....	....	....	....	....	....	....	....
VJ	56	?	?	30	30	?	?	0	0	0	0	0	0	0	0	0	0	0	B	1	2
VJ2	37	21	19	21	19	?	0	0	0	0	0	½	0	0	0	0	0	0	B	1	1
IVG7	22	0	0	0	0	0	0	0	3	12	good	3	12	good	0	0	0(?)	not tested	B	2	1
VA9	21	3(?)	3(?)	0	0	0	0	0	3	12	good	3	12*	good	0	1	0	0	B	2	2
VD	56	0	0	0	0	0	0	0	4	12	good	4	14	good	0	0	0	0	D	1	1
VIID	55	?	?	0	0	0	0	0	4	6	good	4	6	good	0	0	0	0	D	0	0
IVE1	10	1	1	0	0	0	0	0	3	5	good	3	5	good	1	1	0	0	B	....	....
IVF	44	0	0	0	0	0	0	0	5	7	good	0*	2	poor	1	1	0	0	D	2	2
VA1	38	?	?	0	0	0	0	0	3	9	good	2	8	good	1	1	0	0	D	1	1

† Weeks, ‡ Months; * Impacted cerumen.

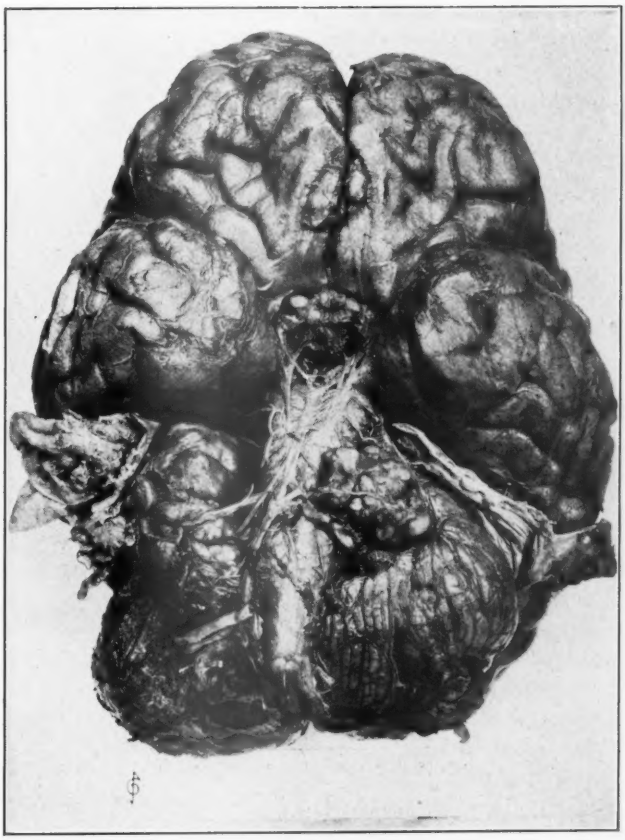

Fig. 1. Patient VA6. The larger tumor on the right is attached to the dura.

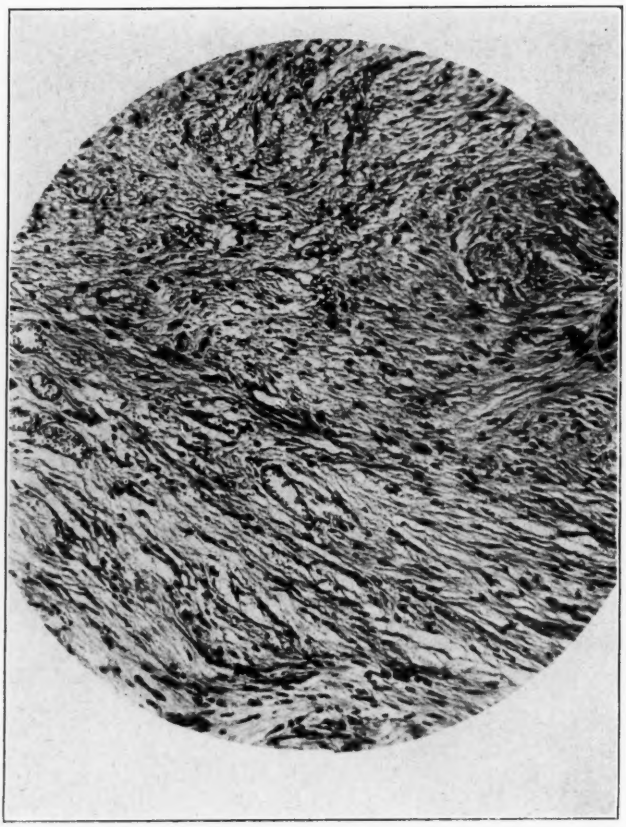

Fig. 2. Patient VA6. Fibrous streams with interspersed areas of looser reticular nature. A suggestion of palisading is evident to the right of the center. Hematoxylin and eosin stain; ×160.

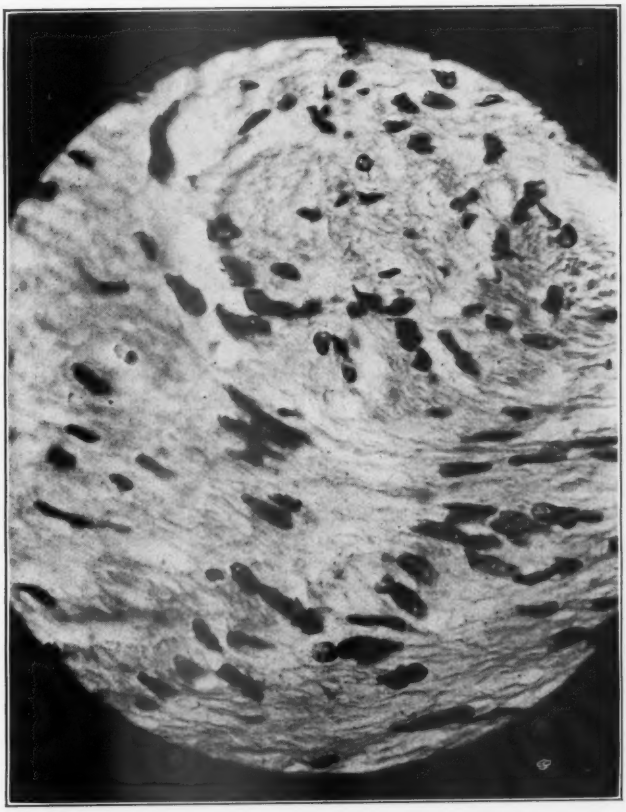

Fig. 3. The same field shown in Fig. 2, but under a higher power; ×627.

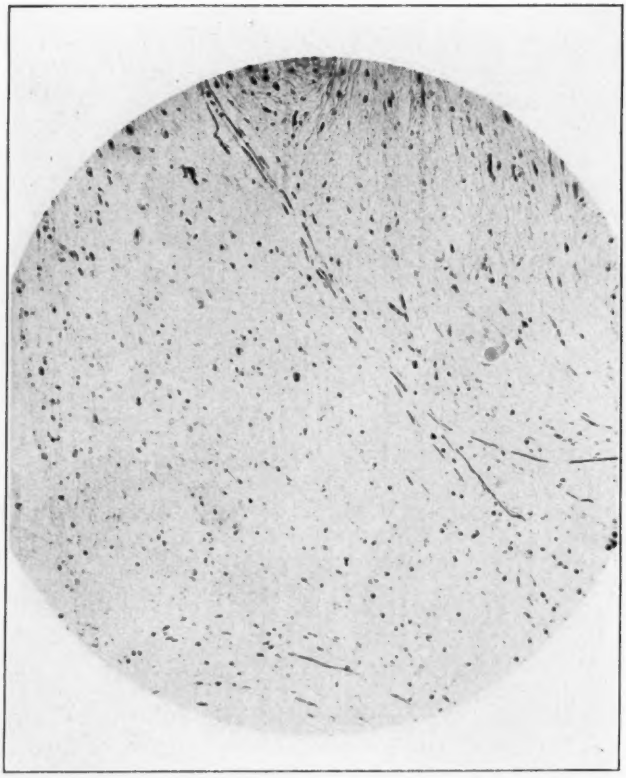

Fig. 4. Patient VA6. Nerve fibres are seen invading the tumor tissue. Gross-Bielschowsky stain; ×160.

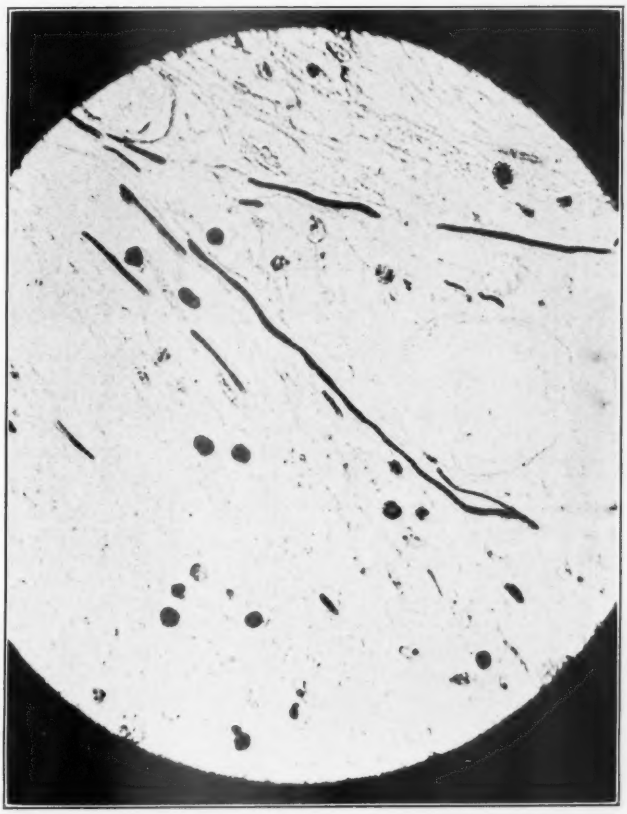

Fig. 5. The same field shown in Fig. 4, but under a higher power; ×627.

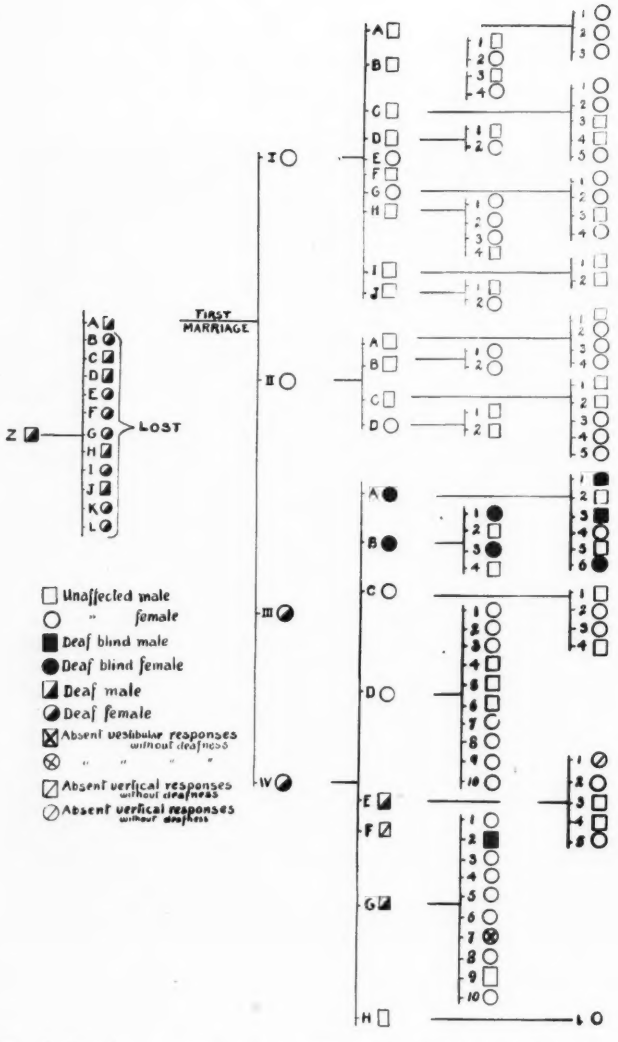

Fig. 6. The first generation is represented by Z, the second by A to L, the third by Roman numerals, the fourth by capital letters, and the fifth by figures. The fifth generation consists of a double column. For the sake of simplicity, the issue of the two marriages of Z A are separated.

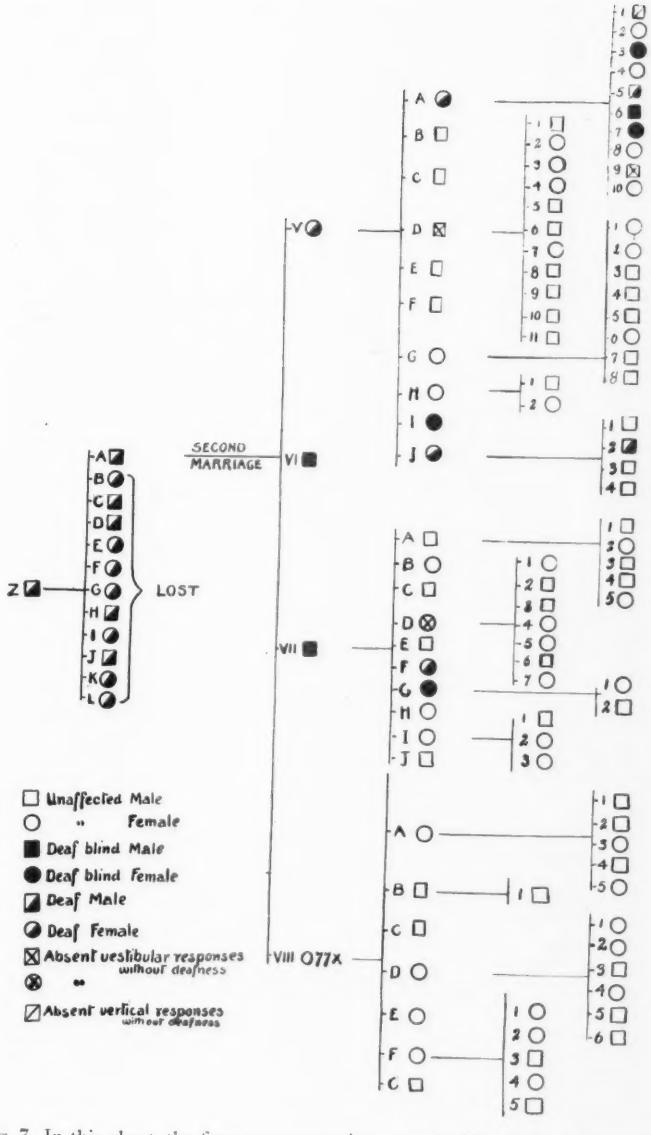

Fig. 7. In this chart, the first two generations seen in Fig. 6 are duplicated.

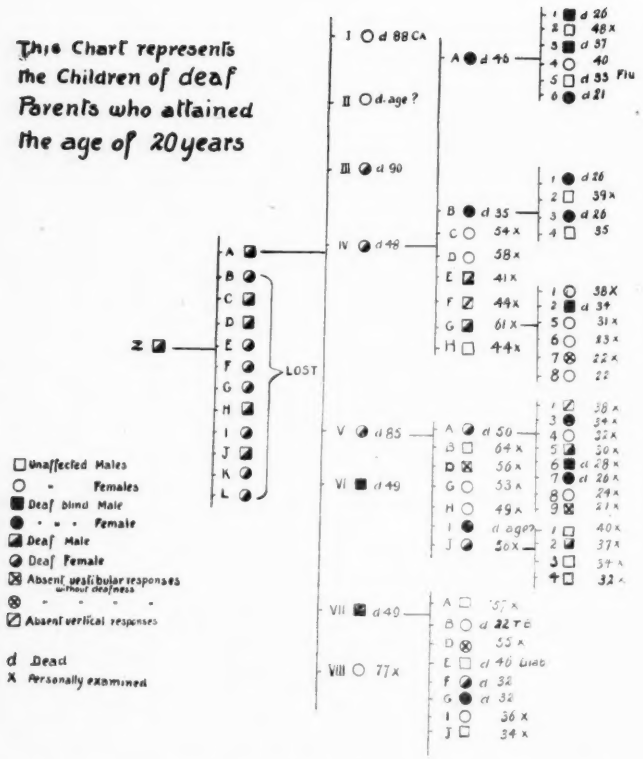

Fig. 8. This chart shows the children of deaf parents who attained the age of twenty years. Only the children of affected parents were affected. The average age at onset of deafness was twenty years.

LXIX.

PRIMARY ASEPTIC CAVERNOUS SINUS THROMBOSIS.

GEORGE C. KREUTZ, M. D.,*

DETROIT.

Primary aseptic cavernous sinus thrombosis is a clinical rarity Friedenwald[1] reports such a condition in a woman of sixty-three and bases his diagnosis on bilateral exophthalmos, conjunctival edema and intraocular changes. There was consecutive involvement of the intraocular structures, but symptoms finally subsided with a typical picture of thrombosis of the central veins with final atrophy of the disc. His patient had nephritis, glycemia and hypotonia. Likewise, there was cerebral arteriosclerosis with temporary attacks of aphasia. In the course of her illness this patient lost consciousness and had convulsions followed by visible increase of exophthalmos and conjunctival edema. The aseptic character of the disease was indicated by absence of fever and by the gradual development and slow disappearance of the condition. Eagleton[2] feels that "local thrombosis of the cavernous sinus and of the lateral sinus, aseptic and protective in nature, is a very frequent occurrence."

The rarity with which reports of this clinical entity appear in the literature warrants the recording of a similar case in which the clinical impression is strongly supported by symptoms and signs pointing toward involvement of the cavernous sinus in an aseptic thrombotic process.

Viola T., age 29, white, married, mother of two male children (fourteen months and five weeks old, respectively), came to the Henry Ford Hospital Outpatient Department on April 20, 1928, with a chief complaint of "sore eyes and headaches." In her history it was developed that about March 1, 1928, the patient developed swelling of the right eyelids and severe right temporal headaches. One week later, she was delivered of a normal male infant. There was no postpartum fever, and patient stated that her recovery, other than the eye condition, was uneventful.

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This, however, did not clear. The swelling of the right lids increased and the right ball itself began to protrude. Four weeks later, or about one week before we saw her, the left eyelids began to swell and she then noticed for the first time that vision in the right eye had decreased considerably. The headaches, likewise, increased and became localized to the right frontal and temporal regions. With this there developed a continuous humming sound over the right temporoparietal region. She had occasional sensations which she described as "chills," but repeated temperature readings were said to be normal. X-rays of her teeth had been obtained and one crowned tooth extracted without any improvement in the eye condition and attending symptoms.

When first examined by the writer, on April 20, 1928, the patient was in evident pain. The right eyeball was markedly proptosed, the eyelids were swollen, the conjunctival and scleral vessels engorged and tortuous. Visual acuity in the right eye was 6/60. She could not move the eyeball in any direction. The pupil was regular, 6 mm. in diameter, reacted slightly to light but not in accommodation. The cornea and iris were normal. The fundus examination showed marked hazing of the disc without any measurable choking, but there was no definite cupping. The veins were very distended and tortuous. There were a few scattered patches of exudate, no hemorrhages. The left eyelids, likewise, were swollen, the conjunctival and scleral vessels engorged, the eyeball somewhat proptosed, but to a lesser degree than the right. The pupil was regular, 4 mm. in diameter, and reacted to light and in accommodation. There was no disturbance in the left extraocular muscles. The fundus picture showed some distention and tortuosity of the veins but no hemorrhages or exudate. There was no papilledema. Visual acuity was 6/15 with difficulty. The patient was admitted to the hospital for further observation and study.

A complete physical examination failed to show much of interest. Heart and lungs were normal. The blood pressure was 112/80 on the right and 104/80 on the left. A neurologic examination did not reveal any pathologic signs other than those above described in the eyes and eye muscles. There was questionable diminution in the tactile sense over the area supplied by the

ophthalmic branch of the fifth nerve. Cardiologists' studies showed no clinical evidence of septicemia. Pelvic examination showed slight relaxation of the pelvic outlet, fundus in good anteposition, no abnormalities in the adnexæ. Nose and thorat examination showed innocent appearing tonsils. The nasal mucous membranes appeared normal by both anterior and posterior rhinoscopy. There was neither clinical nor X-ray evidence of paranasal sinus disease. Repeated tests showed urine to be free of albumin and casts. One specimen showed a trace of sugar, but blood sugar at that time was 91 mgms. per 100 cc. of blood. Repeated complete blood examinations never revealed a leucocytosis. Blood Wassermann tests and blood cultures were negative. Nonprotein nitrogen was 27 mgms. Phenolsulphophthalein test for kidney function showed that the patient excreted 56 per cent of the dye in two hours. The temperature chart during a four day stay in the hospital was flat, and subsequent daily 4 p. m. readings in the home were normal over a period of two weeks. Repeated temperature readings during and immediately after the patient was having her "chilly sensations" were always normal. White blood cell counts, likewise, never varied from normal at these times.

By May 23, 1928, three months after the onset, the condition had reached its height. The right fundus now showed large fresh hemorrhages. The disc was obscured by exudates which likewise were spread throughout the fundus. The left fundus showed marked tortuosity and distention of the veins but no hemorrhage, nor was any hemorrhage ever seen in this fundus. The patient complained mainly of the following symptoms: (1) Terrific headaches associated with a severe humming noise in the right parietal region which could be lessened and sometimes stopped entirely by strongly compressing the right side of the neck over the carotid and internal jugular areas. (2) Marked diplopia with practically no abductor power on the right side, and only sight power on the left. (3) Progressive diminution in vision in both eyes so that the patient could barely see large objects with the right eye, and she was not able to read Jaegar chart No. 7 with the left eye. Both pupils were dilated and fixed to light and accommodation. Treatment up to this time had been entirely

symptomatic. She was requiring frequent use of morphia to control headaches, which were so severe that the patient went to bed with window shades drawn and icebags to the head.

Early in July, 1928, the patient was placed on massive doses of sodium iodide intravenously. Coincident with this medication head noises stopped, but at their cessation the edema of the left eyelids became more pronounced, and the scleral and conjunctival vessels became more engorged. No new findings were present intraocularly, however. She likewise developed severe pains over the bridge of the nose. Following this exacerbation conditions remained unchanged for a period of two months.

Late in September, 1928, the condition began to improve rapidly. The proptosis disappeared almost entirely, lids became soft and wrinkled, and the scleral and conjunctival vessels showed very little engorgement. In the right fundus old blood and exudate had been absorbed, and there was no evidence of fresh hemorrhage. However, vision remained unchanged and the internal strabismus was increased. The right eye now was turned inward and upward so far that half of the iris was not visible. Moderate headaches persisted. Head noises were gone.

By early November, the sixth nerve paresis had begun to subside, so that the patient could move the eyes slightly laterally, the left more than the right. The pupils reacted in a normal manner to light and in attempting to accommodate. In the left eye visual acuity was 6/10 and the patient could accommodate well enough to continue reading a favorite serial story in the daily paper. The fundi showed no venous distention or tortuosity, and there was no hemorrhage or exudate. The right disc was rather pale. Headaches had disappeared except for occasional flare-ups which were easily controlled by small doses of acetyl salicylic acid. She had assumed the full responsibility for her household duties and was eating and sleeping well without medication.

In May, 1929, visual acuity was 6/60 and Jaeger No. 7 right, and 6/7.5 and Jaeger No. 1 left, without glasses. Abductor power was improving steadily, left more than right. There was no swelling of the lids and no headaches. Improvement continued and by November, 1929, the muscle power of the left eye was normal, but there was a definite right convergent strabismus with

only slight abductor power. Visual fields showed peripheral vision only on the right with a large central scotoma. The left field was not abnormal. The right fundus showed a central choroiditis and pale disc. The left was not abnormal. An advancement of the right external rectus after the method of Worth and partial internal rectus tenotomy was done under local anesthesia, with fairly good cosmetic results. Visual acuity in the left eye was 6/6 with sphere minus 0.50, cylinder plus 0.75, axis 75 degrees. Vision in the right eye could not be improved with a corrective glass. It is now two and one-half years since the onset of this patient's illness and she is in excellent health. She became pregnant in March, 1930, but aborted spontaneously without untoward systemic results.

CONCLUSIONS.

The absolute diagnosis in a case presenting the history, physical signs and symptoms above enumerated will, in the absence of an opportunity to examine the brain stem and cavernous sinus directly, always remain obscure. Yet, because of its aseptic character, its unilateral origin, its subsequent extension to the opposite orbit, the progressive involvement of the fifth, sixth and third nerves, and the evidence of obstruction to venous return from the orbit and fundi, and gradual resolution, the clinical impression of an aseptic thrombotic process in the cavernous sinus seems justified.

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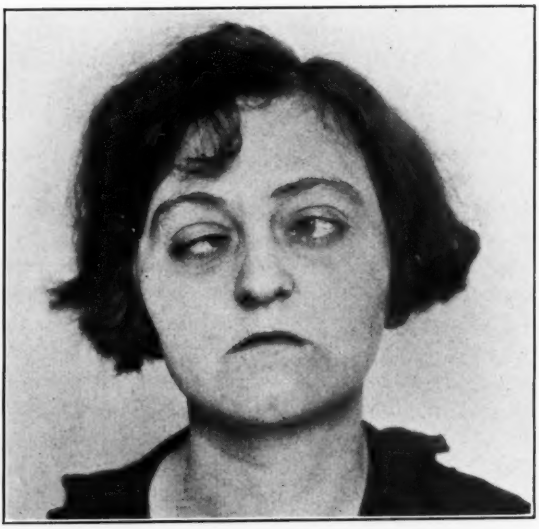

Fig. 1. Taken April, 1928.

Fig. 2. Taken November, 1929.

Fig. 3. Taken April, 1928.

Fig. 4. Taken November, 1929.

LXX.

THE SUPPURATION OF THE PETROUS PYRAMID: PATHOLOGY, SYMPTOMATOLOGY AND SURGICAL TREATMENT.*

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AND

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NEW YORK.

PART I.

INTRODUCTION.

ANATOMIC AND PATHOLOGIC DATA.

INTRODUCTION.

We have concerned ourselves with the problems presented by otitic meningitis for many years. In the course of our studies it became apparent that, in the interval of time which elapses in most cases between the otitic and mastoid lesion and the therapy applicable to them, on the one hand, and the terminal lesion in the meninges, on the other, there ensues a period during which the patient is almost free from symptoms. This penultimate period would ordinarily pass unnoticed. Only after the conclusion of the case, in the postmortem scrutiny of symptoms, pathologic data and operative findings, did the lesser signs of that interval assume significance. Finally, once a relationship was established between the penultimate phase of the infection and the terminal meningitis, and diagnostic aid was developed by means of roentgenograms of the petrosal pyramid during life and before the onset of the terminal meningitis, diagnosis of the lesion in the petrous pyramid became more exact and a distinct

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clinical entity was evolved which is the topic of this and our succeeding papers.

Before proceeding with the details of our observations, it is necessary to enunciate certain fundamentals to clarify some prevalent conceptions. Considerable confusion exists regarding the so-called Gradenigo syndrome—the pathologic changes underlying those cases which present trifacial pain and abducens paralysis, sometimes accompanied, but more often unaccompanied by changes in the cell content of the cerebrospinal fluid. In the studies which follow we are not dealing with the so-called Gradenigo syndrome, except incidentally. We hold with Vogel[1] that the series of events which are usually characterized by the term Gradenigo syndrome are not characteristic of a specific lesion and do not form a distinct clinical entity. The uncritical acceptance of the idea that a distinct clinical entity formed the basis of the Gradenigo syndrome is one of the reasons for the confusion which exists with regard to the real significance of suppuration of the petrosal pyramid.

Gradenigo,[15] in his original studies, brought forward no histologic evidence to support his belief that the triad of symptoms he described are diagnostic of any one organic pathologic condition. Panse[2] disagrees with Gradenigo, stating that the triad does not furnish the typical picture of a disease. Boonacker and Huizinga[3] find that the otogenous abducens palsy appears only in the winter months and is accompanied by other evidences of grippe and hence is a toxic neuritis. Geronzi[4] believes that the Gradenigo syndrome is either reflex or toxic in nature. Körner[5] states that abducens palsy has no diagnostic significance, since it is often found in mild intracranial complications of otitic suppuration which heal spontaneously. Alt[6] gives six etiologic factors for the causation of the Gradenigo syndrome; and Köllner[7] and Sears[8] take the same view. Lange[9] states that when widespread abscesses in the immediate vicinity of the abducens cause no paralysis of that nerve, one cannot take the stand that there is more than a casual relationship. Uffenorde,[10] in 1929, repeats the opinion he expressed in 1920, that while the Gradenigo triad is very seldom found in petrosal tip suppuration, trigeminal irritability is very often evident; and he further states that one

should depend much less on the abducens palsy as evidence of perilabyrinthine suppurative foci. Vogel sums up the arguments against the triad as representative of a given pathologic condition by stating that "when a symptom complex is beclouded by other factors or varies, it no longer is a symptom complex characteristic of a certain condition. Otogenic paralysis of the abducens is not diagnostic of affections of the pyramidal tip, and in itself does not furnish indications for extensive operative procedures on the pyramidal tip."

In order to make what follows comprehensible, it is necessary to reiterate our belief in the Wittmaack[11] theory of pneumatization of the mastoid process, supported as it is by histologic, roentgenographic and clinical observations. It is on this work that we base our explanation of the mechanics of invasion of the petrosal pyramid, of the symptomatology which follows invasion of the perilabyrinthine air cells and of the causation of chronicity in middle ear infections which, occurring later than early infancy, have their onset in an acute middle ear infection in a fully pneumatized temporal bone.

It is our purpose in these papers to show the anatomic basis of the lesion under discussion, to describe its specific pathology, symptomatology and clinical course and to propose a technic for surgical therapy to deal with it, so that its advance into the endocranial cavity may be arrested. In illustration of our points, we shall report a series of cases wherein we diagnosed the lesion exactly and applied the surgical measures which we are presenting, with recovery in the majority of the cases which were operated upon.

Suppurations of the petrosal pyramid are of two varieties: (a) frank suppurations of the pyramid, more particularly its tip, and (b) osteomyelitis of the pyramid. In this and the immediately succeeding papers we shall confine ourselves to the first group of lesions. While osteomyelitis of the petrosal pyramid ultimately leads to the endocranial structures, its route of advance is not as specific, it does not form as marked a clinical entity in its development and the same surgical technic is not applicable to it. We shall have occasion to refer to this type of lesion in passing, reserving detailed consideration of it for an-

other communication. It is of rarer occurrence than the lesion giving frank suppuration in the petrosal pyramid, and it happens to occur in diploetic rather than pneumatized bone.

Finally, in view of the co-authorship of these papers, it is desired at this point to give exclusive credit for the development of the surgical operation to combat the lesion to Dr. Ralph Almour, who worked out the technic on cadavers.

ANATOMIC AND PATHOLOGIC DATA.

Anatomy.—In the discussion of this problem we are not concerned with fundamental anatomic data. While certain of these will be taken up in the section devoted to the therapy of the disease, at present only those factors will be dealt with which elucidate its pathogenesis and symptomatology.

Normally, the tympanic cavity is transformed immediately after birth from a space filled with embryonal connective tissue into one containing air. During the process of development, which begins soon after birth, the infantile mastoid process—a spongy bone containing marrow between its trabeculæ—is subjected to the pneumatizing influences of the tympanic mucosa. In brief, the process of pneumatization consists of an ingrowth of the subepithelial embryonal connective tissue into the marrow spaces which communicate with the air-containing antrum. This connective tissue displaces the marrow originally contained between the bony trabeculæ. With the increasing age of the individual, the embryonal tissue begins to undergo a certain amount of contraction and fibrosis so that it becomes contracted down toward the bony walls. In this process it draws with it the epithelial lining, first from the antrum into the neighboring cells and then from the latter into other cells adjoining them, and so on. As the subepithelial tissue contracts still further, it eventually reaches its adult structure as a thin layer of connective tissue stroma closely applied to the trabeculæ. Covering this is the epithelial lining. Thus a series of air-containing chambers is created from formerly marrow-containing spaces, and these communicate with the middle ear through the antrum (Wittmaack).

Examination of a well pneumatized temporal bone discloses the fact that pneumatic cells are by no means limited to the mastoid portion of the bone. They are seen to occupy the squama and the zygoma also. They are found in the floor of the middle ear, under the tegmen and more extensively around the mouth

Fig. 1. Vertical section through antrum of seven months old child. The first, second and third stages of pneumatization are seen. At points marked X, the connective tissue has invaded the spaces previously filled with marrow, a few of which are still visible at the upper portion of the field. At AC, the contraction of the connective tissue is almost completed and an air-cell has formed. At A the antrum is seen fully pneumatized. (Almour.)

of the eustachian tube. It thus becomes evident that wherever there are marrow spaces which are in contact with tympanic mucosa the latter can exert its pneumatizing influence and convert them into air-containing chambers, which will then communicate directly with the middle ear.

From an embryologic standpoint, the petrous bone in reality consists of two distinct bones. One, the otic, or labyrinthine capsule, houses the membranous labyrinth and conforms to its contour and windings. It, in turn, consists of three distinct layers of bone. The innermost—the one immediately bounding the membranous labyrinth—is termed the endosteal layer and, as its name implies, is derived from the connective tissue endosteum which everywhere lines the perilymphatic space. The intermediate or

Fig. 2. An extensively pneumatized temporal bone. Note cells in squama, floor and roof of middle ear, and around mouth of eustachian tube.

endochondral layer is developed from cartilage and shows—what is peculiar to this layer—a retention of islands and strands of cartilage throughout life. These two layers are compact bone and have reached their full size at birth. The outer or periosteal layer is connective tissue bone derived from the periosteum. That portion of it which forms the inner tympanic wall comes in contact with the tympanic mucosa and, in its developmental stage, may be subjected to the pneumatizing power of that

tissue. It therefore happens that very often the periosteal layer shows definite tracts of pneumatic spaces which surround the other layers. Siebenmann[12] described four tracts of cells which extended from the antrum under the superior semicircular canal to run along the posterosuperior surface of the petrosa, eventually

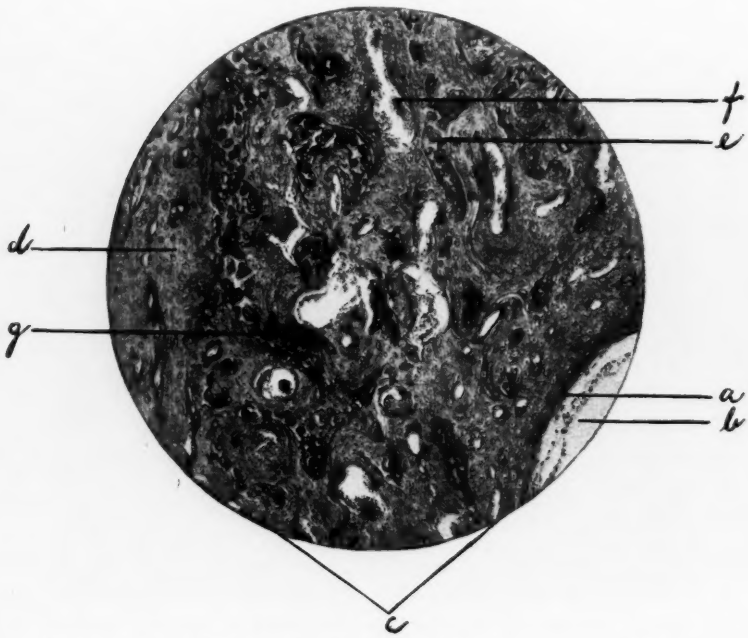

Fig. 3. Vertical section through labyrinth of one-year-old child, in vicinity of vestibule. Low power. Hematoxylin-eosin.
a—Endosteal layer; b—Vestibule; c—Endochondral layer; d—Periosteal layer; e—Interglobular space showing calcified cartilage ground substance and unopened cartilage cells; f—Blood vessel; g—Large strand of calcified cartilage—large cartilage rest. (Almour.)

coalescing at the apex of the bone. In Lange's two cases, he microscopically traced a tract of cells which began at the mesial antral wall and ran above and behind the superior canal to reach the petrosal tip finally. Uffenorde states that in well pneumatized bones he has observed a large cell which starts from the antrum

and passes deep into the pyramid behind the superior canal, and a second cell extending from the epitympanic space inward and running parallel to the first. He has observed a third group of cells which go in a retrolabyrinthine direction behind the posterior canal and occupy Trautmann's space. Eagleton,[13] in his studies on the cadaver, in three out of nine cases found tracts of cells situated in the perilabyrinth in the periosteal layer which led from the region of the superior semicircular canal directly to the petrous tip.

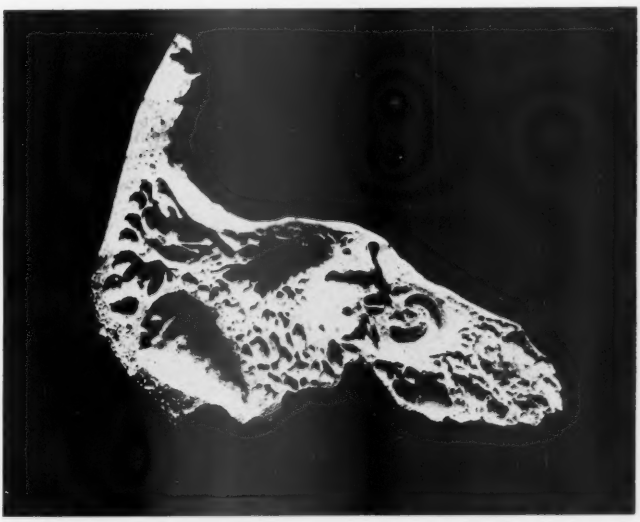

Fig. 4. A temporal bone cut across the labyrinth, to show the extensive pneumatization of the petrosal tip.

The remainder of the petrous bone, excluding the otic capsule, is normally a spongy bone containing marrow spaces. However, if subjected to the pneumatizing influence of the tympanic mucosa, either by way of the peritubal cells or via the perilabyrinthine cells, it also can become converted into a bone consisting of air-containing spaces. Various observers have reported the

finding of large pneumatic spaces in this portion of the petrous bone.

Von Troeltsch[14] noted that very often pneumatic cells were not confined to the mastoid process but were seen to broaden out and sometimes involve the entire pyramid. Merkel[16] also described the frequent occurrence of pneumatic spaces within the pyramid, resulting in a pneumatization of more than one-half of the petrosa. Urbantschitsch[17] states that he has often seen small bony foramina in the vicinity of the tubal ostium and in the upper posterior half of the inner tympanic wall, through which openings a probe could be passed into pneumatic spaces well forward in the tip of the petrous pyramid. In one instance the probe could be visualized through the thin bony superior wall of the pyramid.

Baldenweck[18] describes a petrous bone in which the tip was composed of two large cells superimposed one upon the other. Girard[19] describes a chain of cells extending from the peritubal area into the petrosal tip as far as the foramen spinosum of the sphenoid. Collet[20] describes a specimen in which the petrous tip contained a cell measuring 12 mm. in its long axis and 10 mm. in its transverse diameter. Surrounding this cell and communicating with it were numerous smaller cells which extended forward and backward. Mangebeira-Albernaz[21] describes a temporal bone whose pyramid showed a large cell 29 mm. long, 14 mm. high and 9 mm. broad; the entire pyramid was 48 mm. long. It is thus seen that in well pneumatized temporal bones pneumatic spaces may be present in the perilabyrinthine area and the petrosal tip as well as in the mastoid bone.

Other anatomic factors of importance in the comprehension of the lesion are the relationships of the various soft tissue structures which come in contact with or pass through the temporal bone. These are as follows: The membranous labyrinth: the 5th, 6th, 7th, 8th, 9th, 10th and 11th nerves; the carotid artery; the eustachian tube; the petrosal nerves.

The Membranous Labyrinth.—This structure is housed in the bony otic capsule, which is always seen as a compact bone. No reports have as yet appeared to show that pneumatization ever occurs in the inner two layers of this bony capsule. Consequently

it is protected from invasion at all points except the natural openings into it, viz., the oval and round window, the endolymphatic and cochlear aqueducts and the internal auditory meatus. An involvement of the perilabyrinthine cells or of those in the petrosal tip may therefore cause an irritation of the membranous labyrinth, but not an inflammatory disease, unless erosion occurs into one of the preformed channels.

The Fifth (Trigeminus) Nerve.—The relationship of this nerve to the petrous portion of the temporal bone is of the utmost importance in understanding the symptomatology of the lesion. The fifth nerve, both motor and sensory roots, after arising from the pons, passes forward and, through an oval opening in the dura above the internal auditory meatus, reaches the superior border of the petrous bone. It then runs between the bone and the dura, along the superior surface of the petrosa, to the apex of the pyramid, where the sensory root takes the form of a large semilunar ganglion, known as the Gasserian ganglion. This lies in a depression on the upper surface of the petrous apex which, with the overlying dura to which the ganglion is firmly adherent, forms a pocket for its reception. Underneath the ganglion lies the great superficial petrosal nerve. From this ganglion three large branches are given off—the ophthalmic, the superior maxillary and the inferior maxillary.

It is with the ophthalmic branch of the fifth nerve that we are most concerned. Within the cavum Meckeli, the ophthalmic branch has a longer course than the other two divisions. In this area it is firmly bound down and can be separated only with difficulty from the overlying dura and from the superior surface of the petrosa to which it is attached. In addition it is closely adherent to the cavernous sinus. The second and third branches of the fifth, on the other hand, are not bound down in their course. In the ganglion the fibers rest on the carotid artery and the branches are not adherent to the dura. Consequently, any disturbance of the position of the Gasserian ganglion will cause tension to be exerted on the ophthalmic division first, while the other two branches, because of their flexibility, can accommodate themselves for a time to their new position (Eagleton).

The Sixth (Abducens) Nerve.—The dissections of this nerve by Dorello,[22] Vail,[23] Wheeler[24] and lately by Eagleton have given us an accurate idea of its relationship to the petrous bone. After leaving the lower border of the pons, the nerve runs upward and outward to pierce the dura over the sphenoid bone. It then turns forward and passes between the apex of the petrous bone and

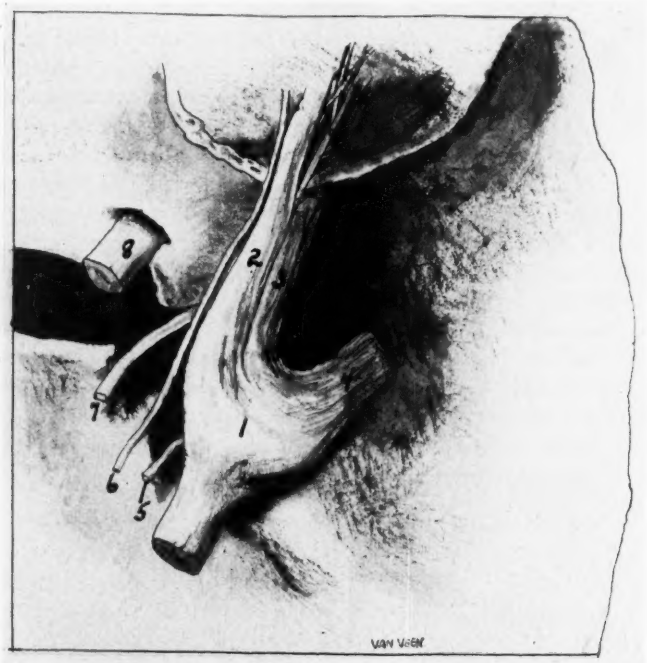

Fig 5. Semidiagrammatic sketch of Gasserian ganglion. 1. Gasserian ganglion; 2, ophthalmic root; 3, maxillary root; 4, mandibular root; 5, abducens nerve; 6, trochlear nerve; 7, oculo-motor nerve; 8, optic nerve.

the posterior clinoid process of the sphenoid. The space between these two bony structures is formed into a canal by the interposition of a very strong fibrous bundle known as Gruber's ligament, or the petrosphenoidal ligament. This ligament forms, with the upper margin of the petrous bone, a three cornered space, in

which lie the superior petrosal sinus and the abducens nerve. This canal has been termed Dorello's canal.

Eagleton's dissections of this area show differences in the length of the nerve in this canal in different specimens. In addition, the nerve is sometimes found tightly constricted and immobilized at this point, whereas in other specimens it is fairly loose and mobile. Eagleton has determined also that Dorello's canal varies as regards position, shape and size, all of these factors being determined by the anatomic structure of the bony basis cranii.

The Seventh (Facial) Nerve.—This enters the internal auditory meatus with the eighth nerve, lying at first to the inner side of the latter and then above it. At the depth of the meatus it enters the fallopian canal, running outward to reach the inner tympanic wall, where it forms a reddish swelling known as the geniculate ganglion. At this point the large and small superficial petrosal nerves are given off, the former to connect with the sphenopalatine ganglion and the latter with the otic ganglion. It also gives off, at this point, the external superficial petrosal nerve, which communicates with the sympathetic filaments accompanying the middle meningeal artery. The remainder of the course of the seventh nerve is within the fallopian canal, until it emerges from the stylomastoid foramen, where it is distributed to the muscles of the face. The chorda tympani nerve is intimately associated with the facial from a quarter of an inch above the stylomastoid foramen backward into the medulla.

The Eighth (Acoustic) Nerve.—This enters the internal auditory meatus and divides into its cochlear and vestibular portions, to be distributed respectively to the spiral and Scarpa's ganglia.

The Ninth (Glossopharyngeal) Nerve.—This nerve leaves the skull at the central portion of the jugular foramen and is enclosed in a separate sheath of dura and arachnoid. It occupies a position anterior to the eleventh and twelfth nerves. In its passage through the jugular foramen it forms a groove on the lower portion of the petrous portion of the temporal bone. It is the sensory nerve of the pharynx, fauces and tonsil; it is the motor nerve of the pharyngeal muscles and a special sensory nerve of taste to the posterior third of the tongue. Within the

jugular foramen it presents two ganglionic enlargements: the upper is called the jugular ganglion and the inferior is the petrosal ganglion. The latter sends communicating branches to the vagus and the sympathetic. It also gives rise to Jacobson's nerve, which enters a bony canal in the under surface of the petrosa and then the middle ear, where it forms the tympanic plexus. From here branches go to the round and oval windows and the lining of the eustachian tube and tympanum. The tympanic plexus sends communicating branches also to the carotid plexus, the great deep petrosal nerve and a nerve which runs through the petrous bone to form, with a branch from the facial, the lesser superficial petrosal nerve.

The Tenth (Vagus) Nerve.—This is both a motor and a sensory nerve. It emerges from the skull through the jugular foramen accompanied by the spinal accessory, the two occupying a position posterior to the glossopharyngeal, from which they are separated by a membranous septum. It has a wide distribution, supplying the larynx, heart, lungs, esophagus and stomach. It receives communicating filaments from the seventh, ninth and eleventh nerves.

The Eleventh (Spinal Accessory) Nerve.—In the course of this nerve, both its spinal and accessory portions accompany the vagus in its passage through the jugular foramen. It supplies motor filaments to the trapezius muscle and to the sternomastoid. Its accessory portion joins the vagus.

The Petrosal Nerves.—As above described, these arise from the facial and glossopharyngeal nerves. The former gives rise to the great superficial petrosal, the latter to the small superficial petrosal. The great superficial joins with the deep petrosal from the carotid plexus and both run in grooves situated on the superior surface of the petrous bone. They eventually join to form the vidian nerve, which enters the sphenopalatine ganglion, and so communicates with the superior maxillary branch of the fifth nerve. The lesser superficial petrosal passes through the petrous bone to enter the otic ganglion. The latter is situated immediately below the foramen ovale and lies on the inner surface of the inferior maxillary nerve. Internally it is in relationship with the cartilagious portion of the eustachian tube.

The Internal Carotid Artery.—This enters the carotid canal in the petrous bone, runs upward for a short distance, then curves forward and inward, and again ascends as it enters the cranial cavity. At first it lies below and in front of the cochlea and mid-

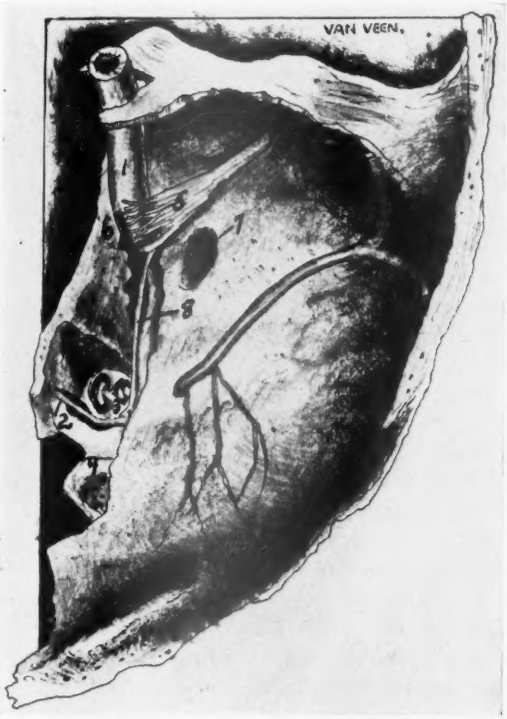

Fig. 6. A temporal bone viewed from above with Gasserian ganglion lifted aside, and bony carotid canal opened. The cochlear and the superior semi-circular canal exposed. 1, carotid artery; 2, facial nerve; 3, cochlea; 4, superior semi-circular canal; 5, Gasserian ganglion; 6, pyramidal tip; 7, foramen ovale; 8, great superficial petrosal nerve.

dle ear, then behind and internal to the eustachian tube. Further on it is separated from the Gasserian ganglion by a thin plate of bone, which often is absent and replaced by fibrous tissue.

Pathology.—From the cases reported in the literature, from our own personal cases and from macroscopic and microscopic examinations, there is unquestionably a distinct pathologic entity which we term suppuration of the petrosal tip.

The avenues of infection are:

1. From the antrum or epitympanic space, above or below the superior semicircular canal, following the posterosuperior surface of the petrosal into the pyramidal tip.
2. From the peritubal cells into the pyramidal tip.
3. From the peritubal cells directly into the carotid canal or through dehiscences in the anterior tympanic wall into the carotid canal and then rupturing into the cavum Meckeli.

Most of the observations in regard to the avenues of infection are macroscopic and tend to favor the peritubal cells and the carotid canal as the avenues of invasion. However, the microscopic studies of cases of petrosal tip suppuration show the pathway of infection to be along the perilabyrinthine cells originating in the antrum and the epitympanic space.

The finding of Urbantschitsch,[17] in one of his specimens, demonstrates that a definite connection exists between the peritubal cells and the petrosal tip. He injected fluid into the opening of one of the peritubal cells, and this fluid filled the pyramidal tip and leaked out into the middle cranial fossa. Hilgermann[25] traced the infection in the tip from cells around the tube and floor of the middle ear, between the jugular fossa and the labyrinth, to end in the pyramidal tip. Other observers, among them Uffenorde,[26] Wheeler, Bürkner,[27] Druss and Friesner,[28] all mention this route of infection.

Microscopic evidence, however, seems to disprove the peritubal cells as the avenue of invasion and establishes the perilabyrinthine route as the path of infection. Lange,[9] in a microscopic study of two cases of pyramidal tip suppuration, found identical pathologic conditions in both instances. In the vicinity of the petrous tip was a large abscess bordering on the dura. From this area the serial sections demonstrated the tract of infection to proceed along the cells above and behind the superior semicircular canal, which led directly to the mesial antral wall. The peritubal cells in the two cases were uninvolved and showed no connection with

the suppurative focus in the petrous tip. Lange is of the opinion that petrosal tip suppurations have their origin in the antrum and extend along the tract described, and that in those instances wherein the peritubal cells were noted to be involved the tubal area represented the avenue of escape of the purulency. We are inclined to accept this view as a logical one and will offer clinical proof of it later.

In the cases of Friesner and Druss, the first case showed the extension of the infection from the antrum to the cells in the perilabyrinthine area along the posterior margin of the superior canal and thence to the pyramidal tip. The second case likewise showed this route of infection; but in this instance an egress for the pus was established in the region of the internal auditory meatus, so that the petrous tip showed inflammatory involvement to a lesser extent than in the first case.

Styx[29] was the first one to discuss the route to the petrosal tip via the carotid canal. Sheppard,[30] Elkund[31] and Brunner have also described this route in their cases.

The pathologic picture in all cases studied postmortem shows an extension of the purulent focus in the middle ear into the petrous tip through one of the channels above mentioned. Uffenorde found pus in the petrous pyramid in all his cases, with marked purulent infiltration of the Gasserian ganglion. Von Troeltsch[32] found two extradural abscesses on the upper surface of the petrosa, which connected with a pus cavity in the pyramid through a perforation in the bone. Ostmann's[33] case showed pus filling the entire petrous pyramid up to the tip with a rupture through the superior surface involving the Gasserian ganglion. Grünert's[34] three cases all showed suppurative foci in the pyramid which led to sequestration and the formation of extradural abscesses. Habermann's[35] patient succumbed to a meningitis due to an osteomyelitis of the region of the antrum and eustachian tube which extended into the pars petrosa. Leutert[36] reported a subdural abcess at the tip of the pyramid located between the foramen lacerum and the Gasserian ganglion, wherein the entire petrous tip was transformed into a sequestrum. Lange's two cases also showed large abscesses in the petrous tip. Elkund's case showed a large abscess of the pyramidal tip with large cells

filled with pus around the opening of the eustachian tube. Wilkinson's[37] case at autopsy showed an enormous abscess cavity in the petrosal tip. Eagleton's first case showed a carious process of the petrous apex which had eroded both the anterior and posterior surfaces of the pyramid. A large cavity was present in the pyramid, filled with granulation tissue and pus. The Gasserian ganglion was edematous. His second case showed a large cavity in the petrous apex, filled with cheesy pus and detritus. An erosion into the middle cranial fossa occurred which caused the Gasserian ganglion to be surrounded by granulation tissue and to be edematous. The route of invasion in this case was through the supratubal cells. Friesner and Druss record similar findings of purulency within the petrous pyramid.

The above findings are in cases wherein a purulent process within the petrous pyramid extended into the cranial cavity and resulted in death. In all of these cases it is evident that an original focus within the middle ear eventually spread to involve not only the mastoid process but also the petrosal cellular tissue and eventually resulted in exitus of the patients, due to a rupture into the endocranium. From a clinical observation of nine cases of petrosal tip suppuration, five of which have recovered and are still living, we are prepared to add to the postmortem findings several additional factors which can be observed during the progress of the case from a simple otitic suppuration to its final stage in the petrosal tip.

1. All of our cases had an extensively pneumatized mastoid process. At the primary operation the cells extended well forward into the zygoma. In the region of the antrum and in the area posterior to the superior semicircular canal, cellular elements were present.

2. As the suppuration in the mastoid process and middle ear clears up, the suppurative process spreads into the perilabyrinthine tracts toward the pyramid.

3. After a period during which the middle ear remains dry, there suddenly reappears a profuse aural discharge, as a source of which the mastoid wound can be definitely ruled out, for it appears healthy and contains no pus. This finding leads us to believe with Lange that the pus within the petrous pyramid finds

an avenue of escape by rupturing through the cells around the eustachian tube and then out through the middle ear.

The latter observation adds an additional factor to our knowledge of the pathology of chronic aural suppuration. Wittmaack's studies on pneumatization, both normal and pathologic, have demonstrated that infantile otitis media hyperplastica (that originally described by Aschoff[38] as due to meconium, vomitus, etc.) causes a cessation of the normal process of pneumatization at the end of the first stage. The embryonal subepithelial connective tissue grows into the marrow spaces of the mastoid process; but, due to the continued irritation set up by the foreign-body otitis media, contraction of the myxomatous tissue does not occur as in the normal process of pneumatization (Wittmaack and Almour). Consequently there remains within the intratrabecular spaces of the mastoid process this connective tissue, which persists and eventually undergoes metaplastic bone changes. The result of this process is a sclerotic mastoid, which lasts throughout life. Wittmaack,[39] Eckert-Möbius,[40] Mannasse[41] and recently Almour[42] have shown that this type of faulty pneumatization, namely, interference with the normal contraction of the myxomatous subepithelial tissue in the mastoid process, predisposes to chronicity of suppurative lesions of the middle ear. This, together with the röle that the squamous epithelium plays in the production of chronic aural suppurations, accounts for the greatest percentage of cases of chronic purulent otitis media.

We accept Wittmaack's work as the only logical explanation of hitherto unexplained observations in cases of chronic otorrhea. Nevertheless, his work does not account for the presence of a chronic suppurative otitis media associated with a normally pneumatized mastoid process wherein the squamous epithelium plays no rôle in either the cause or the cure of the lesion.

In our cases of suppuration of the petrous pyramid, we have been impressed by the recurrence of a discharge from the middle ear after the mastoid wound had completely healed and after the middle ear had been dry for a time. In the cases upon which we have operated for petrosal tip suppuration and which recovered, two still present a chronic aural discharge. The radical cavity is completely epidermatized, there is no rhinopharyngeal

suppuration, inflation does not blow any discharge into the cavity from the tube, and yet a discharge can be seen coming from the region of the tubal ostium at the site of our entrance into the pyramid. In one of our cases, which cleared up without operation, a persistent discharge is present with a completely healed mastoid process. In the section on symptomatology, the clinical significance of this will be discussed further. Here we present an additional pathologic factor in petrosal tip suppurations which has a close relationship to the interpretation of protracted aural discharge in the presence of a pneumatic mastoid process. Uffenorde states that with a protracted acute middle ear infection after operation, one must think of a pyramidal tip suppuration.

(To be continued.)

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LXXI.

BACTERIOLOGIC DIFFERENTIATION AND SPECIFIC ETIOLOGY* OF MASTOIDITIS.

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WITH AN INTRODUCTORY NOTE BY

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Introductory.

Otologists are wont, after diagnosis of otitic lesions, to institute surgical therapy and expect a good recovery in proportion to the timeliness of the intervention and the thoroughness of the surgical procedures. Study of cases giving almost identical symptoms and submitted to identical surgical procedures, and even given grossly almost identical operative field findings eventuate in absolutely different outcomes. One case recovers, the other courses through a series of complicating penultimate lesions and ultimately dies, while the third, its acute phase subsiding, carries on intermittently as a chronic lesion. In the usual study of our cases we are at a loss scientifically to comprehend wherein lie the factors which differentiate the one case from the other. With the usual laboratory aids at command, the study of the bacterial flora obtained from the ear discharge, and from the mastoid process at the time of operation failed to supply this differentiating data. The answer always was whichever prototype of case was studied, that the bacterial invader was the streptococcus hemolyticus.

The study herewith, made by Dr. Hadjopoulos, sheds light not only upon a factor which strongly differentiates one streptococcus from another of the same morphologic group, but it opens a new field for investigation into studies which are hoped will give us

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means to increase the patient's immunity to those graver types of streptococcic infections, and enhance the results obtained by properly applied surgery to the primary, penultimate and even ultimate lesion.

To be successful, in each case, the study differentiating the sub-forms of the streptococci present in a given case must be completed in the varying interval of time which elapses between the purulent otitis media and its complicating sequelæ. And if an immunizing agent is perfected it will have to be administered during this interval; for when the complicating lesion is developed the bacterial flora by that time has already reached its specific characteristics.

Finally, there is necessary a word on the nomenclature used by Dr. Hadjopoulos, where he speaks of the catarrhal type of infection, he means an inflammatory reaction to bacterial invasion limited to the mucosal lining of the middle ear and its adnexa. This should not be confused with the so-called catarrhal otitis with its symptom picture resulting from mechanical or physical reactions from the eustachian tube.

Bacteriologic Differentiation and Specific Etiology of Mastoiditis.

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The specific relationship of streptococci to mastoid inflammations is peculiar. Unlike acute infections whose etiology is known, as typhoid, pneumonia, diphtheria, cerebrospinal meningitis, or even like those of unknown etiology like measles, influenza, poliomyelitis, the various clinical manifestations of mastoiditis are not in themselves the evidence of a clinical entity but are rather a conglomerate of symptoms, and do not furnish the picture of a primary disease. Inflammations of the mastoid process resemble more certain surgical conditions which occur in the course of an infection, as gonorrheal arthritis, as a complication of gonorrheal infection, a tuberculous lesion of the bone happening in the course of a generalized tuberculosis, gangrene

of the toes during the course of diabetes, and traumatic septicemias with secondary localizations.

The reason why mastoiditis is considered a primary disease is because of the still disputed etiology of the precursor diseases as ordinary catarrhal colds, influenza, measles, etc. The relationship of otitis media to mastoiditis is essentially an anatomic one, the true origin of infection being limited to the mucosa of the upper respiratory tract. A careful analysis of the history of mastoiditis would reveal almost invariably the pre-existence of a "cold" or influenza or some accidental traumatism of the upper respiratory mucosa.

In a previous survey of the field of streptococcic septicemias I have pointed to the diversity of the streptococcic flora in relation to mastoiditis. Our studies, then limited to the small number of eleven postmastoidal septicemias, revealed at least three different types. Since then we have undertaken a careful bacteriological study of all mastoid infections in the otologic service of the hospital, and to date our list includes nearly 100 studies.

My technic consisted in inoculating nutrient glucose broth with a swab dipped in the infected mastoid area obtained during the operation. Growth invariably occurred within 24 hours. A loopful of this primary growth was smeared on a plain nutrient agar blood plate. Similarly the differential sugars of the Holman classification were inoculated. The findings are classified according to the hemolytic and the sugar fermenting properties, and also in relation to the various clinical characteristics of the disease as shown in our table. Before we enter into the discussion of my findings, in order to explain certain intricacies of the pathogenesis of mastoid infections it is advisable to repeat certain facts on the relation of the oxygen supply to the growth and the focal selectivity of streptococci.*

Histologically the tympanic and the mastoid cavities constitute a blind sac extension of the nasopharyngeal cavity. The intervening long and narrow eustachian tube affords a continuity to the

*On the importance of focal streptococcic selectivity our readers are referred to the exhaustive studies of E. C. Rosenow and a recent article by the author on the significance of the classification of foci in respect to oxygen tension.

mucosa entirely of endodermal origin. The internal ear is dissociated from the rest in being of ectodermal origin and lined with mucosa formed from it. Consequently a study of the nasopharyngeal bacteriology naturally leads into the course of events relative to the middle ear and mastoid infections, via the eustachian tube.

In terms of oxygen requirements, the streptococcic flora of the nasopharynx come under the following three types: The obligate aerobic (Anginosus-Salivarius, Subacidus-Ignavus) with an incidence of 58 per cent, the facultative aerobic (Pyogenes-Mitis) 21 per cent and the facultative anaerobic (Infrequens-Fecalis) 6 per cent. The remaining 15 per cent is variably divided between eight rather rare and mostly nonpathogenic types, none of which appears in our list of mastoid infections. Under pathologic conditions of the nasopharynx there is a reversal of percentage in favor of the more pathogenic facultative aerobic types, the Pyogenes-Mitis group.

In making a tabulation of the incidence rate of the various streptococci entering into the pathogenicity of mastoiditis, I observed a similar preponderance of the common pathogenic Pyogenes type with 64 per cent, next to which are the strictly aerobic types, Anginosus Salivarius, etc., with 23 per cent, and finally the facultative aerobics with an incidence of 77 per cent. (Table No. 2.)

Therefore, it is more natural to consider otitis media and mastoiditis in the light of a complication to a pre-existing nasopharyngeal infection rather than a disease per se. A careful investigation of the history records of our hospital cases points invariably to the pre-existence of some common respiratory infection like the ordinary cold, influenza, tonsillitis, etc. This substantiates my viewpoint. Under such abnormal circumstances any one of the nasopharyngeal streptococcic flora can invade this blind sac, the natural pathway being the middle ear via the eustachian tube. But once this pathway is invaded a train of differential symptomatology ensues that makes possible a clinical classification of the disease in terms of the particular invading streptococcic type of organism presented.

OBLIGATE AEROBES: ANGINOSUS-SALIVARIUS.

Due to their absolute dependence on the free supply of oxygen, their pathogenicity (Anginosus, mainly) is strictly limited to the exposed mucosa of the upper alimentary and respiratory tracts. The inflammation is invariably of the acute catarrhal, congestive or granular and at times even of the hemorrhagic and necrotic types. If the initial inflammation does not subside naturally within a week or ten days, the chances of its spread into the middle ear through the eustachian tube increase. In this new environment, in all respects similar to the original focus, the inflammation for a short while runs its typical course of an acute catarrhal otitis media, manifested by a turgent red and slightly bulging tympanic membrane accompanied by moderate pain. The inflammatory products naturally are of the acute catarrhal type —i. e., a mucoserosanguineous fluid of moderate amount. In the meantime certain changes occur which alter this natural course. The one of prime importance is the closure of the eustachian opening due to the inflammatory edema. The whole cavity, originally an open one with sufficient oxygen, now becomes temporarily closed with a complete limitation of oxygen supply to the oxyhemoglobin of the turgent mucosa and the serosanguineous exudate. Hereafter there is an accumulation of the exudate, more pressure, therefore more pain, and bulging of the tympanic membrane. With no surgical intervention the outcome would depend on the degree of accumulated immunity. If this is sufficient it is also aided by the gradual exhaustion of oxygen supply to render the cavity less adapted to the needs of the invading host and thus would lead either to a temporary or even to a permanent recession of the inflammation and to recovery. Usually, however, there occurs a spontaneous rupture of the drum membrane, resulting in partial release of pressure, abatement of pain, lowering of temperature, and, in fact, to signs leading to recovery unless there is a clogging of the artificial opening. At this stage we reach to the second phase of the disease, which leads invariably to hospitalization and eventually to surgical intervention.

During the short interval during which there is discharge from the external auditory meatus, the streptococcus acquires new

virulence due to its access to a fresh supply of oxygen. The discharge, probably due to external contaminants, becomes slightly purulent, fills the cavity and gradually seeps through the antrum into the mastoid process with its mass of interconnected cells. A new train of symptomatology develops now, which usually starts our hospital records. A gradually rising temperature, which may be of a moderately septic type; possibly some seropurulent discharge from the ear; a definite postauricular tenderness, etc. The cellular structure of the mastoid, by this time devoid of air and partly devitalized, undergoes a hemorrhagic necrotic change, with its spaces becoming filled with a moderate amount of thinly purulent serosanguineous exudate. Eventually the process becomes a sac of necrotic tissue still preserving the cells' outlines with its trabeculæ. Surgical intervention is usually reached at this stage with invariably successful results. The mortality in this type of infection is next to nil unless there be a preexisting serious complicating cause. In the case of delayed surgical intervention the necrotic process may extend to the immediate neighborhood, giving rise to cortical perforation, sinus wall granulation, dural and sinus plate necrosis and not very uncommonly a transient septicemia. In very young children, where the formation of the antrum and cellularization of the mastoid is not yet complete, the infection runs an uninterrupted continuous course involving both middle ear and mastoid almost simultaneously, and there usually results a cortical perforation with subperiosteal abscess or necrosis formation.

The hospital records of a hypothetical case would read as follows: The patient, a male or female, of an age around 5 to 10 (Anginosus) or middle age (Salivarius) gives a history of an ordinary cold or grippe, possibly with a sore throat and cough a week or ten days prior to admission. Within four to five days, the patient experiences first a sensation of clogging and later an intermittent shooting pain in one of the ears. Following a restless night the parents notice a moderate elevation of temperature and a physician is called who diagnoses the case as one of otitis media catarrhalis acuta. A paracentesis is performed, yielding a slight serosanguineous fluid. For the following day or two the patient feels more comfortable, the temperature gradually falls

to almost normal level, the pain eases considerably and the discharge becomes more and more scant till it stops almost completely. Thereafter (two or three days prior to admission) all symptoms return, more intense, causing hospitalization. On admission, a fever of 102° or thereabout, pain in the affected ear, and a definite postauricular tenderness clinches the diagnosis. The X-rays disclose a slight or moderate diminution of illumination of the affected mastoid area, some thinning or complete atrophic absorption of the intercellular structure. Of the other laboratory findings the hematology shows a moderate leucocytosis of 12,000 to 13,000 on an average, and the case is operated upon with such pathologic findings as enumerated before. The subsequent course is usually an uneventful one, ending in complete recovery within two to three weeks.

FACULTATIVE AEROBES: PYOGENES-MITIS.

This very important and highly pathogenic group, because of its certain well defined biologic characteristics, can be readily differentiated from the Anginosus-Salivarius group in terms of clinical symptomatology. In respect to their oxygen requirements, both members of this group are facultative aerobes and are capable of surviving under abnormally low oxygen tensions. Their metabolic requirements are derived from fermentation of simple carbohydrates as well as the splitting of complex proteins. Unlike the Anginosus group when implanted on otherwise healthy mucous membranes, they can penetrate into deeper structures, invariably locating in the submucosa and stroma in close proximity to the lymphatic, vascular and nervous plexuses. Thus located, by means of their positive chemiotaxis, they invite active leucocytosis which ultimately results in painful deep seated pyogenic infections requiring surgical intervention, viz., tonsillar and retropharyngeal abscesses, recurrent paranasal sinusitis, surgical infections, etc.

Usually, the production of sufficient local immunity on the part of the host and timely surgical intervention can abort their further spread. Half-way means, on the other hand, result invariably in increasing their virulence and in the invasion of lymphatics, with an acute lymphangitis and toxic degeneration of the

glands that drain the area and later even a pyogenic lymphadenitis, leading to a fatal septicemia.

The involvement of the middle ear and the mastoid process by this group of microorganisms is generally traceable to a pyogenic infection of the nasopharynx. An analysis of the records in the great majority of cases reveals some severe infection of the upper respiratory tract in the form of influenza, or recurrent colds accompanied by any one or more of the following complications: Coughs, sore throats, tonsillitis, sinusitis—these are found to the extent of 70 per cent. Or a history of pneumonia, whooping cough, parotitis combined, 17 per cent, and finally the recurrence of a pre-existing middle ear or even a partly healed mastoid condition, 15 per cent.

The spread of the nasopharyngeal infection is not solely limited to the continuity of the lining mucosa, as in the former class of catarrhal infections, but can follow the course of the lymphatics and the blood stream. It may thus involve such areas as the jugular bulb, causing a primary jugular thrombosis without even an otitis or mastoiditis, although an otitis is more often present than absent.

The nature of the infection is strictly pyogenic, giving rise to a high leucocytosis. The average leucocytic count in our list of 58 cases was 18,000, against 13,000 of the catarrhal group. Coincident with the high leucocytosis there is an increase in the percentage of the segmented forms, 77 per cent, against 74 per cent.

The onset of the disease is characterized by a sudden, intense, throbbing earache, an intermittent fever notably of the septic type and a gradual accumulation of the purulent exudate in the middle ear. On account of the steadily increasing pressure and the accumulation of the proteolytic enzymes of leucocytes as well as of the inciting micro-organisms, a spontaneous rupture of the drum membrane occurs before the family physician is called in. An otoscopy before the rupture would therefore reveal a white, distended tympanum whose puncture would give rise to a profuse thick and purulent discharge. Whether the rupture be spontaneous or by paracentesis, it is usually followed by considerable

relief of the majority of symptoms. Nevertheless, the mastoid process is invariably involved. The air cells eventually are all broken down and a markedly septic turn ensues in the course of the disease, thus necessitating hospitalization.

On admission, the patient, a male or female, child or adult, is toxic, having a septic temperature which ranges between 99 and 104 degrees, sometimes as high as 105 or 106 degrees. The previous history and the existing signs and symptoms invariably lead to a correct diagnosis and prompt surgical intervention. The value, therefore, of laboratory data becomes of minor significance in aiding diagnosis, but the laboratory is of paramount importance in differentiating this new type of a pyogenic mastoidal infection, with its very high mortality rate, from the previous described far less dangerous form, the catarrhal necrotic type.

In this respect the X-rays disclose a marked decrease in illumination of the mastoid process with an almost complete disappearance of cellular structure, a condition usually diagnosed by the roentgenologist as destructive mastoiditis. In the previous group it was a slight or moderate diminution of illumination, accompanied by thinning and absorption of cellular structure, diagnosed as necrotic mastoiditis. The hematologic picture comes next as a differential aid. The leucocytosis is invariably higher than that of Anginosus-Salivarius; it averages around 18,000 and runs as high as 39,000, against an average of 13,000 with the Anginosus group. The differential leucocytic values in both types show a proportional increase of the segmented forms, while the unsegmented staff forms are more abundant in this class.

The post-operative findings demonstrate the far reaching destructive potentialities of this highly pathogenic group of microorganisms. The type of complications, therefore, comprises an area much wider than that of middle ear and mastoid process proper. We enumerate here the most frequent; subperiosteal abscesses, cortical perforations, dural and epidural abscesses, perisinus abscess, sinus plate perforations, sinus phlebitis, sinus and jugular thrombosis, meningitis, cerebellar and cerebral abscesses, septicemias and finally remote metastatic abscesses in muscle sheaths and bone marrow. A further unfortunate feature of this

type of mastoid infection is its association with chronic debilitating diseases, such as diabetes, myo- and endocarditis, bronchopneumonia and erysipelas.

With timely intervention the most serious of the above complications may be avoided. Nevertheless, this group takes the highest toll from among its victims. The mortality rate in our list of 58 cases was 35 per cent. In conclusion, the early differentiation of this type of infection is very important, as it means early surgical intervention, the only bar in the destructive course of these highly pathogenic micro-organisms.

FACULTATIVE ANAEROBES: INFREQUENS-FECALIS.

The natural habitat of these streptococci is the human intestinal tract. Under normal circumstances they represent 85 per cent of the fecal streptococcic flora. When pathogenically inclined they usually give rise to chronic inflammations. Although by nature anaerobic they can live and still preserve their pathogenicity under strictly aerobic conditions.

In the upper respiratory and alimentary tracts they can be isolated from such partly or totally closed and chronically infected foci as the gums, dental caries, tooth roots, nasal accessory sinuses, etc. Some of these biologic characteristics were witnessed in the records of our cases—four out of six (history of seventh case missing) were chronic sufferers of a previous otitis or mastoid infection in association with a still active paranasal sinusitis. The number of our cases belonging to this group are, however, too small for generalizations. In particular, the course of infection is usually a protracted one, occurring in children under ten and giving rise to a moderately septic temperature. The complications are of the nature of chronic infections with granulation tissue covering the affected areas. There was no fatality in this group.

THE PNEUMOCOCCUS GROUP: STREPTOCOCCUS MUCOSUS CAPSULATUS

Of the various pneumococci, type III is the one invariably encountered in middle ear and mastoid infections. It is essentially an encapsulated streptococcus but does not display any constant

class characteristics as outlined under the previous groups. Unlike true streptococci, the mucosus capsulatus gives very little symptomatology at the onset of the disease. The gravity of the infection is realized towards its close, which is mostly fatal. In our list of five cases three died with brain abscess. Of the surviving two, one was a pneumococcus type I. It is fortunate that the incidence of this group is very low.

In Table 2 we sum up the various class characteristic features of streptococci entering into the etiology and pathogenesis of mastoid intections.

TABLE I.

A CLINICAL AND BACTERIOLOGICAL ANALYSIS OF NINETY-ONE CASES OF MASTOIDITIS.

A. THE OBLIGATIVE AEROBIC GROUP:

ANGINOSUS-SALIVARIUS

Laboratory Number	Admission Number	Duration of Disease in Days	Age of Patient	Fever: Highest	Fever: Course	Leucocytic Count and Differential: Highest Count Preop. in 1000	Leucocytic Count and Differential: Average Count of 4 Days Postoperative: W. B. C. in 1000	Leucocytic Count and Differential: Average Count of 4 Days Postoperative: Differen. Segm'ted %	Leucocytic Count and Differential: Average Count of 4 Days Postoperative: Staff Forms %	Hemolytic Values	Sugars Fermented: Mannite	Sugars Fermented: Salicin	Sugars Fermented: Lactose	Holman Classification	Diagnosis Post-operative: Acute: Catarrhal (1)	Acute: Haemorrhagic (2)	Acute: Necrotic (3)	Acute: Purulent-Coalescent (4)	Acute: Chronic Recurrent (1)	Acute: Labyrinthitis (1)	Acute: Primary Jug. Thromb. (2)	COMPLICATIONS: By Contiguity: Subperiosteal Abscess (1)	By Contiguity: Cortical Perforation (2)	By Contiguity: Dural Pl. Necrosis (3)	By Contiguity: Perisinus Abscess (4)	By Contiguity: Sinus Pl. Granulation (5)	By Contiguity: Sinus Pl. Necrosis (6)	By Contiguity: Sinus Phlebitis (7)	By Contiguity: Sinus Thrombosis (8)	By Contiguity: Meningitis (9)	By Contiguity: Brain Abscess (10)	Remote: Metastatic Abscess (1)	Remote: Erysipelas (2)	Remote: Broncho Pneumonia (3)	Remote: Diabetes (4)	Remote: Myo-Endocarditis (5)	Outcome
46	33451	31	10	102	SL	....	18	....	4	D	—	—	+	Ang				*																			
56	34279	5	2	102		....	....	....	....	B	—	—	+	"			*							*													Rec
69	34801	18	32	105	SL	8	8	76	9	B	—	—	+	"			*							*													"
24	26023	43	30	104	SL	15	11	80	15	B	—	—	+	"					*					*													"
51	33861	5	1	101		....	....	....	....	B	—	—	+	"			*							*													"
26	32087	6	2	104	SM	21	18	73	10	B	—	—	+	"			*	*						*													"
70	34997	9	4	101		....	....	....	....	B	—	—	+	"				*						*													"
71	35071	5	2	101		20	....	83	....	B	—	—	+	"				*						*													"
73	35300	13	7	104		18	14	76	9	B	—	—	+	"				*						*													"
7	28914	33	5	103	SM	25	18	74	20	B	—	—	+	"		*												*	*	*							"
8	28792	14	43	102		17	12	76	18	B	—	—	+	"				*																			"
9	29068	20	2	103	SM	19	10	80	18	B	—	—	+	"					*	*																	"
30	32747	13	8	104	SM	19	19	73	11	B	—	—	+	"		*	*																					"
77	35508	7	7	103	SL	14	13	84	7	B	—	—	+	"				*																			"
12							10	78		B	—	—	+	"																								"
6	28927	9	1	102	SL	18	13	60	5	A	—	—	+	Sal				*				*															"

																	1	2	3	4	1	1	2	1	2	3	4	5	6	7	8	9	10	1	2	3	4	5	
37	33259	9	46	100		....	9	....	....	A	—	—	+	"				*							*														"
58	34374	7	39	101	SL	12	9	69	6	A	—	—	+	"					*	*				*	*			*											"
12	22509	15	2	104		17	12	65	10	A	—	—	+	"						*																			"
20	30952	10	7	105	SM	15	12	68	7	A	—	—	+	"	*																								"
25	31950	17	45	100		16	14	70	10	A	—	—	+	"					*	*																			"

B. THE FACULTATIVE ANAEROBIC GROUP:
INFREQUENS-FECALIS

																	1	2	3	4	1	1	2	1	2	3	4	5	6	7	8	9	10	1	2	3	4	5	
5	28719	24	8	104	SL	26	18	82	6	B	+	+	+	Inf						*	*							*												Rec.
19	30393	7	25	100		12	9	72	9	B	+	+	+	"						*						*	*	*												"
43	33587	39	7	106	SH	23	17	72	14	B	+	+	+	"					*		*							*	*		*									"
2	28583	56	2	102	SL	20	18	82	9	B	+	+	+	"						*	*																			"
3	28700	17	2	101		14	11	50	8	B	+	+	+	"						*	*																			"
4										B	+	+	+	"																									"
11	29526	8	1	104		13	12	52	8	A	+	+	+	Fec										*	*															"

C. THE PNEUMOCOCCUS GROUP:

																	1	2	3	4	1	1	2	1	2	3	4	5	6	7	8	9	10	1	2	3	4	5	
17	30680	51	53	100		11	9	77	10	A′	—	+	+	III						*	*	*										*	*							
22	31516	43	22	102	T	34	12	79	33	A	—	+	+	III						*		*				*	*	*						*						Died
28	32324	20	68	106	T	22	20	93	25	A′	—	—	+	III						*	*					*	*	*	*	*		*	*	*						"
14	29586	10	49	99		13	13	79	15	A	—	—	+	III							*								*											Rec.
29	32761	10	2	104	SM	24	24	76	20	A′	—	+	+	III							*								*											"

D. THE FACULTATIVE AEROBIC GROUP:
PYOGENES-MITIS

																	1	2	3	4	1	1	2	1	2	3	4	5	6	7	8	9	10	1	2	3	4	5	
10	29114	7	60	105	T	29	21	89	48	B	—	+	+	Pyo						*	*					*	*	*												
21	31214	36	42	100		26	20	....	11	B	—	+	+	"								*												*				*	*	Died
23	31782	17	8	107	SH	26	19	62	47	B	—	+	+	"										*							*	*								"
33	32062	9	5	106	SM	30	26	86	12	B	—	+	+	"									*									*								"
36	33190	17	7	105	SH	38	19	86	14	B	—	+	+	"							*				*	*			*			*	*							"
45	33099	42	14	105	SM	37	14	84	12	B	—	+	+	"					*									*					*	*						"
47	33209	43	.....	105	SH	30	29	83	18	B	—	+	+	"					*	*									*		*				*		*			"
50	33796	18	3	105	SH	39	24	84	12	B	—	+	+	"					*						*			*	*		*	*	*							"
62	34494	10	3	107	SH	22	16	80	28	B	—	+	+	"					*	*							*				*			*						"
64	34506									B	—	+	+	"																										"
66	34798	6	13	108	SH	38	24	80	11	B	—	+	+	"						*	*	*						*				*								"

TABLE I—Continued.

Laboratory Number	Admission Number	Duration of Disease in Days	Age of Patient	Fever: Highest	Fever: Course	Leucocytic Count and Differential: Highest Count Preop. in 1000	Leucocytic Count and Differential: Average Count of 4 Days Postoperative: W. B. C. in 1000	Leucocytic Count and Differential: Average Count of 4 Days Postoperative: Differen. Segm'ted %	Leucocytic Count and Differential: Average Count of 4 Days Postoperative: Staff Forms %	Sugars Fermented: Hemolytic Values	Sugars Fermented: Mannite	Sugars Fermented: Salicin	Sugars Fermented: Lactose	Holman Classification	Diagnosis Post-operative: Acute: Catarrhal	Acute: Haemorrhagic	Acute: Necrotic	Acute: Pur-Confescent	Acute: Chronic Recurrent	Acute: Labyrinthitis	Acute: Primary Jug. Thromb.	By Contiguity: Subperiosteal Abscess	By Contiguity: Cortical Perforation	By Contiguity: Dural Pl. Necrosis	By Contiguity: Perisinus Abscess	By Contiguity: Sinus Pl. Granulation	By Contiguity: Sinus Pl. Necrosis	By Contiguity: Sinus Phlebitis	By Contiguity: Sinus Thrombosis	By Contiguity: Meningitis	By Contiguity: Brain Abscess	Remote: Metastatic Abscess	Remote: Erysipelas	Remote: Bronchopneumonia	Remote: Diabetes	Remote: Myo-Endocarditis	Outcome
															1	2	3	4	1	1	2	1	2	3	4	5	6	7	8	9	10	1	2	3	4	5	
72	35118	6	4	106	SH	24	28	83	6	D	—	+	+	..				*	*											*							
74	36074	40	7	106	SH	34	22	82	6	D	—	+	+	..			*	*	*											*							Died
75	34912	31	7	106	SH	25	24	86	12	B	—	+	+	..				*	*						*					*							..
80	35703	18	10	105	SH	27	15	78	8	B	—	+	+	..					*											*							..
4	18091	2	25	107	T	22	22	91	....	B	—	+	+	..				*	*											*	*						..
13	15550	14	6	104	SH	32	26	77	....	B	—	+	+	..				*	*											*							..
22										B	—	+	+	..																*	*						..
25	26691	149	23	106	SH	24	19	84	20	B	—	+	+	..			*	*	*													*						..
28	27464	5	8	106	T	28	15	70	10	B	—	+	+	..			*	*	*																			..
15	29594	43	16	106	SH	20	15	83	40	B	—	+	+	..					*																			..
53	34009	14	38	101						B	—	+	+	..				*							*													Rec.
54	34119	21	28	101	SL	18	14	80	6	B	—	+	+	..				*	*																			..
17	23989	19	8	103	SL	12	8	60	8	B	—	+	+	..				*	*						*													..
27	32088	24	7	104	SM	28	23	81	34	B	—	+	+	..			*	*	*																			..
32	32985	16	6	105	SM	33	18	82	17	B	—	+	+	..		*	*	*	*				*		*													..
1	22582	8	6	102		23	21	80	7	B	—	+	+	..				*	*																			..
13	29585	6	21	100		18	13	72	12	B	—	+	+	..				*	*																			..
16	30152	11	8	102		18	15	79	8	B	—	+	+	..				*	*																			..
18	30359	11	63	105	SM	19	15	85	6	B	—	+	+	..				*	*																		*	..

																1	2	3	4	1	1	2	1	2	3	4	5	6	7	8	9	10	1	2	3	4	5	
40	33304	7	2	102	SL	22	15	69	10	D	—	+	+	"				*		*							*	*										"
44	33615	8	42	100		....	10	60	5	D	—	+	+	"				*						*			*											"
49	33793	30	7	104	SM	15	12	77	35	B	—	+	+	"				*	*									*										"
55	34111	15	3	106	SM	27	24	80	12	B	—	+	+	"				*	*									*										"
61	34504	10	14	102	SL	....	19	77	9	B	—	+	+	"				*	*					*			*	*										"
78	35911	5	6	101		....	....	....	....	B	—	+	+	"					*	*	*							*										"
79	35864	44	4	102		15	13	63	5	B	—	+	+	"					*		*						*											"
14	15911	8	18	103		16	16	71	....	B	—	+	+	"					*	*			*		*	*	*											"
31	32992	6	4	102		....	....	....	....	B	—	+	+	"					*					*															"
38	33267	11	1	104	SL	....	14	....	7	D	—	+	+	"				*					*	*															"
41	33315	6	3	101		18	16	....	8	B	—	+	+	"				*						*															"
42	33450	4	3	104		....	22	....	10	D	—	+	+	"				*						*															"
57	34298	10	5	102		19	16	72	5	B	—	+	+	"				*	*					*															"
65	34512	11	2	103	SM	24	16	70	7	B	—	+	+	"					*				*																"
76	35488	5	3	101		....	16	....	6	B	—	+	+	"				*	*					*															"
27	27148	50	6	106	SH	16	11	80	20	B	—	+	+	"		*		*			*								*	*				*				"
35	33117	7	3	102		....	19	59	8	D	—	+	+	"				*			*																	"
39	33290									B	—	+	+	"																									"
48	33768	10	2	101	SL	....	17	67	5	B	—	+	+	"						*																			"
52	33996	9	3	101		23	21	80	9	B	—	+	+	"				*	*																				"
59	34471	8	25	103	T	....	30	87	10	B	—	+	+	"				*	*																				"
60	34468	19	12	103	T	24	21	81	8	B	—	+	+	"				*	*																				"
63	34531	6	2	103	T	14	11	71	6	B	—	+	+	"					*																				"
34	33081	4	6	101		....	15	....	8	B	—	+	+	"						*																			"
24	31591	14	46	102	SM	15	15	80	12	A	—	+	+	Mit					*									*										"
67	34659	6	9	101		....	21	79	3	A′	—	+	+	"					*				*																"
68	34559	17	7	104	SM	13	12	76	5	A	—	+	+	"							*		*																"
8	21684	50	9	107	SH	10	8	70	3	A	—	+	+	"		*	*	*																					"

NOTES: T—toxic; SL—septic low; SM—septic moderate; SH—septic highly; *—indicates the presence of described complication. Ang—Anginosus; Sal—Salvarius; Inf—Infrequens; Fec—Fecalis; Pyo—Pyogenes; Mit—Mitis; B—Beta Hemolytic; D—Delta Hemolytic; A—Viridans; A′—Alpha Prime Viridans.

TABLE II.

THE BACTERIOLOGIC CLASSIFICATION OF MASTOIDITIS AND ITS DIFFERENTIAL FEATURES.

Streptococcic Groups of Mastoiditis Etiology	Course of Fever	Site of Infection Proper	Type of Inflammation	Incidence		Leucocytosis	Differential		Complications				Mortality	
				Cases	Rate	Average Count of 4 Days Post-Operative	Segmented Forms	Staff Forms	1-7 Inclusive, See Table I	8—Sinus Thrombosis	9—Meningitis	10—Brain Abscess	Cases	Rate
Anginosus	Slightly to Moderately Septic	Mucosa	Acute Catarrhal	15	16%	14,000	78%	12%	.....	.....	.....	.....	....	.....
Salivarius			Hemorrhagic	6	7%	11,000	66%	8%	(12)	.....	.....	.....	....	.....
Group			Necrotic	21	23%	13,000	74%	10%	64%	.....	.....	.....	....	.....
Pyogenes	Moderately to Highly Septic	Mucosa Submucosa Connective Tissue	Acute	54	60%	18,000	77%	13%	.....	.....	.....	.....	20	37%
Mitis			Subacute Recurrent	4	4%	18,000	76%	6%	.....	.....	.....	.....	....	.....
Group			Purulent Coalescent	58	64%	18,000	77%	13%	(40) 61%	(10) 17%	(14) 24%	(7) 12%	20	35%
Infrequens	Slightly to Moderately Septic	Mucosa Submucosa Connective Tissue	Chronic	6	7%	15,000	71%	9%	.....	.....	.....	.....	....	.....
Fecalis			Recurrent	1	1%	12,000	52%	8%	.....	.....	.....	.....	....	.....
Group			Purulent	7	8%	14,000	68%	9%	(4) 60%	(1) 14%	.....	.....	....	.....
Pneumococcus III	Irregularly Toxic	Mucosa	Mixed	4	4%	13,000	82%	20%	.....	.....	.....	.....	3	75%
Pneumococcus I		Submucosa?		1	1%	24,000	76%	21%	.....	.....	.....	.....	....	.....
Group				5	5%	16,000	81%	20%	(4) 80%	(1) 20%	(2) 40%	(3) 60%	3	60%

Total Cases, 91. Average Mortality of all Groups 25%.

NOTE: As the cases quoted in this work do not represent the total number of mastoids admitted to our hospital during the period of investigation the high mortality figure should be taken as of relative and not absolute value. The true mortality rate during the period 1929-1930 was around 12%.

LXXII.

THE BESREDKA THEORY OF IMMUNITY AND ITS POSSIBLE APPLICATION IN OTOLARYNGOLOGY.*

FRANK J. NOVAK, JR., M. D.,

CHICAGO.

The title of this paper originally included the relation of the reticuloendothelial system and the Besredka theory. Upon further consideration it was thought more expedient to contract the subject, limiting it to a presentation of the theory alone with some of its implications, reserving consideration of the extended subject for another paper or, if it is desired, to consider it briefly in the closing discussion.

A new concept of immunity was presented by Professor A. Besredka of the Pasteur Institute about three years ago. Excepting the men who are actively engaged in immunologic studies there has been little, if any, interest in Besredka's work in this country. Clinical application and investigation of this new theory have been neglected almost completely except in France and in some of the other European countries. In America, Besredka's revolutionary idea of immunity has engaged the academic interest of such investigators as Cannon of the University of Chicago, and Frederick P. Gay of Columbia University.

My purpose in presenting this paper is to try to stimulate clinical interest in this new conception of the mechanism of the immune reaction. I do not propose to give my clinical experience because it is too limited to bear any weight.

The early bacteriologists predicted that the future physician would be an immunologist. Today the average otolaryngologist is very skeptical about the value of the various immunologic practices which are available in the management of the ordinary head cold. Vaccine therapy for infections of the upper respiratory tract is generally regarded as of doubtful value. Besredka proposes a modification in the preparation of vaccine and gives

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a radically new method of administering it. I shall try to present a brief outline of this work without presuming to endorse or to criticise it.

The first in the list (chronologically) of the pathogenic bacteria was the anthrax bacillus. In the year 1850 Davaine observed the presence of small filiform, nonmotile bodies in the blood of a sheep dead of anthrax. He believed that these "filiform infusoria" caused the disease, anthrax. He did an enormous amount of experimental work to prove that the anthrax bacillus was the specific cause of anthrax. His work, however, was not accepted until twenty-seven years later. In 1881 Pasteur made his memorable experiment on 24 sheep, 6 cows and one goat. These animals were vaccinated against anthrax. Then all of the animals were inoculated with a virulent culture of anthrax. None of the animals succumbed. Since that time it has been learned, however, that vaccination of the large animals produces solid immunity in only a relatively small percentage. The vaccination is not unattended by considerable danger to the life of the animal. Moreover, it is known to every laboratory worker that it is impossible to produce immunity in the small laboratory animals, particularly in the guinea pig. It is axiomatic that a guinea pig which comes in contact with anthrax is already a dead guinea pig.

Besredka, studying anthrax, found that a guinea pig whose skin was desensitized by applying a wet compress of an anthrax filtrate to the roughly shaven skin of the belly could receive a dose of live, virulent anthrax bacilli with total impunity. In other words, by immunizing the skin of a guinea pig the whole animal becomes solidly immune to anthrax. The injection of either vaccine or filtrate into the blood, subcutaneously, intramuscularly, or giving it by mouth, fails to produce immunity to anthrax in the guinea pig, but injecting a dose of anthrax filtrate intradermally confers upon the animal a solid immunity. A wet compress appears to be as efficient as the injection, provided the skin is roughly shaved, producing a slight abrasion of the skin. In rabbits immunized by Besredka's method a dose of virulent organisms, three thousand times the minimum lethal dose, failed to kill the animal.

In order to prove that the skin of a guinea pig must be infected with the anthrax bacillus in order to produce the disease, the following experiment was done. Some method of introducing the organisms into the animal without contaminating the skin, had to be employed. Injection through the skin does not preclude the possibility of contamination of the skin with the needle. Plotz used glass, gelatine, and collodion capsules, which were filled with virulent cultures of anthrax bacilli. These were imbedded subcutaneously through a small incision which was allowed to heal. These capsules were then broken, liberating the live cultures in the subcutaneous tissues. The animal suffered no harm. The organisms became "inoffensive when they came in contact with the subcutaneous tissue, and isolated from the skin." However, the organisms that remained in the unbroken capsules did not become attenuated even after six days.

Besredka believes that he has shown that in the guinea pig it is necessary to infect the skin with the anthrax bacillus in order to produce anthrax. Secondly, that the buccal, pharyngeal and gastrointestinal mucosa is impervious to this bacterium, provided the mucosa is not injured by a sharp object. In other words, the organism does not penetrate an intact mucosa. Anthrax then is a skin infection per se. All previous efforts to immunize a guinea pig to anthrax have been futile. Injection of the skin itself with a filtrate of an anthrax culture produces a general solid immunity.

This method of immunization received a searching trial in Syria in 1923. The French cavalry patrolling the wheat fields suffered an alarming loss of horses from anthrax. The Pasteur method of vaccination proved dangerous and was given up. Upon using intradermal vaccination of the filtrates the average mortality among the animals was reduced about twenty times. Altogether about nine thousand horses and mules were vaccinated. The director of the veterinary service called this "a success without precedent in the history in antianthrax vaccination in horses."

In 1923 the commission on epidemics of the League of Nations was confronted with an epidemic of bacillary dysentery among the thirty thousand refugees in Greece. The tissue in-

volved being the gastrointestinal mucosa, an attempt was made at vaccination of this tissue by Besredka's method, using a filtrate of cultures of the Shiga Bacillus. The filtrate was ingested. In one group of seven hundred the epidemic stopped "abruptly and completely," upon vaccination of the entire group. Three hundred and forty individuals arrived at Kokinia, where an epidemic was raging. The new arrivals were vaccinated and although they drank the same water as the rest of the camp, among the vaccinated individuals no dysentery appeared. At the camp of Kokinia four hundred cases of dysentery occurred among 4800 refugees. Two-thirds of the 4800 were vaccinated. In this vaccinated group the epidemic stopped immediately and completely. In the unvaccinated group the epidemic continued for many months.

In a paper entitled "Intradermal Injections of Gonococcal Bouillon Filtrate," Corbus and O'Connor say, "in the treatment of eight women with gonorrheal endocervicitis, four of whom originally presented themselves with salpingitis,—all the tubal symptoms subsided promptly after their first injection and three were gonococcus-free after four weeks. In three young girls with vulvovaginitis of specific origin the results were equally promising. Thirty-seven men have been or are under controlled treatment by this method. Nine are apparently cured after no other type of treatment, systemic or local, but must be kept under observation for several more months to satisfy the most rigid requirements.

Now turning to the matter of the staphylococcus and the streptococcus we find that these organisms have a very low antigenic value. It accounts for the inefficacy of staphylococcus and streptococcus vaccines. Comparing these two organisms with the anthrax bacillus Besredka says "they have, however, one characteristic in common, and that is a negative one; namely, their inability to serve as antigens. Besredka says that a filtrate of a streptococcus and staphylococcus culture, which has been heated will, when injected into the skin of an animal or man, produce immunity which is specific against these two organisms. He cited much experimental work and refers to a considerable amount of clinical work in support of his statement.

Concerning the theoretical basis for local immunization Besredka quotes Pasteur on scientific theories in general when he refers to them as "the illusions of the experimentor, which serve as a guide, and which gradually fade as we travel along the road." In the first place the filtrate consists of the medium in which the organisms have been grown; usually broth or broth serum. The living organisms are filtered out. Now if the filtrate is reinnoculated the bacteria will grow but sparingly. After the second filtration a further effort to innoculate the medium fails. The organisms remain viable but do not grow. This second filtrate contains two substances. One is toxic and thermolabile, and the other is nontoxic, and thermostabile. The filtrate is now heated at 100° Centigrade. This heating destroys the first or toxic thermolabile substance, which Besredka calls virus. The remaining, thermostabile substance, he calls antivirus. The filtrate is now ready for use either as wet dressing or for intradermal injection.

There are two general theories of immunity, the humoral theory, and the cellular theory. It is clearly not within the province of this paper to enter upon a discussion of these theories except as the latter differs from the former in a few salient particulars. Besredka believes that antibodies have but a subordinate part in the immune reaction. They are the digestion products of the interaction between bacteria and phagocytes. When a tissue like the skin reacts with an organism like the anthrax bacillus it does so because the skin cells are sensitive to that particular organism, or some of its products. The application of the filtrate or antivirus, as Besredka calls it, to the skin satisfies this affinity. To put it in other words it desensitizes the normally receptive cells of the skin. Now following this desensitization, when a living culture of anthrax is inoculated into the skin, the cells being no longer sensitive, there is no reaction between the invader and the host and consequently there is no reaction of inflammation. The anthrax bacillus is forced into the role of a saprophite and is promptly phagocytosed largely by the macrophages of the reticuloendothelial system, as Cannon of the University of Chicago has shown. The immune bodies which are found in the blood are considered by Besredka

merely as by-products of the immune reaction. He cites the fact that in certain diseases to which the individual succumbs a large quantity of immune bodies are found, and again in other diseases which the individual survives there is an almost total absence of immune bodies in the blood. Therefore, if the mechanism of immunity is due to the formation of immune bodies the two observations mentioned above are inexplicable. It more easily satisfies logical requirements to assume as Besredka does that the immune bodies are but an incidental product, and that their presence or absence, increase or decrease, does not in any way indicate the presence of or the degree of immunity of an individual.

CONCLUSIONS.

The experience of laryngologists with the various vaccines in the treatment of infections of the upper respiratory tract has been unsatisfactory. At the Pasteur Institute under Besredka a new conception of immunity has been worked out. It has had an extensive trial in Europe, both in the laboratory and in the clinic. It deserves extensive clinical investigation here, and I do not know of a more suitable body for that purpose than the Academy. I recommend a careful reading of two books by Besredka: "Local Immunization—Specific Dressings" and "Immunity and Infectious Disease," both published by the Williams and Wilkins Company.

30 N. MICHIGAN AVENUE.

LXXIII.

CHRONIC DIPHTHERIA ORIGINATING IN AND LIMITED TO THE TRACHEA AND BRONCHI.*

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Diphtheritic infection primary to the trachea and bronchi is of relatively uncommon occurrence, though by no means unknown. Diphtheria, chronic in type, is also recognized occasionally, though this, too, must be regarded as unusual. Chronic diphtheria originating in the trachea and limited to that region throughout its course must be regarded as an exceedingly rare condition. No case exactly paralleling the one to be described has appeared in a search covering the literature for the past fifteen years.

In a monograph *Ueber Bronchialdiphtheria*,[1] published by Max Reh, in 1912, several cases are quoted from an article by Hennig, entitled *Ueber chronische Diphtheria*, that have some points of resemblance. In one a three-year-old child had exhibited for two weeks a slight dyspnea with some cough, no reason for which could be found in the pharynx, larynx or trachea. After a time diphtheria appeared in the larynx and tonsils, and in the tracheobronchial system as well. Unfortunately no lung findings had been recorded. Henoch also reported a case of a child who had been intubated, who coughed up a membrane from the deep lung passages fifteen weeks after the removal of the cannula. Reh also relates a case, which came under his own observation, of a young man who entered the Greifwalder Clinic with a tentative diagnosis of pulmonary tuberculosis. The sputum was negative for tubercle bacilli but positive for the diphtheria organism. At the original examination no suspicion of diphtheria was entertained. A week later, however, diphtheria of the pharynx made its appearance, and after this had cleared up under treatment tubercle bacilli were found in the sputum.

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Lynah[2] made a special study of tracheobronchial diphtheria and reported a considerable number of cases, seen for the most part at the Willard Parker Hospital, New York. In 1916, he reported thirty-eight cases, and in two later reports added several further observations. As the Willard Parker Hospital is maintained for cases of contagious disease only and is free, the cases encountered there are likely to be severe in type and the patients seen only when the condition is acute. Lynah says: "The clinical picture of a case of tracheobronchial diphtheria is quite distinctive of this disease. The onset seldom is sudden and the voice never is lost, and aside from an occasional cough and slight stridor, the condition may be readily overlooked and treated as an ordinary catarrhal cold. . . . There is no doubt that many are overlooked, unless a case of membrane is coughed up, or the larynx becomes involved with a typical picture of laryngeal 'croup.' It is only then that a correct diagnosis is made. There is no doubt that many of the so-called asthmatic attacks which terminate fatally are due to diphtheritic bronchial obstruction. . . . . Any slowly progressive dyspnea should be looked upon with a considerable degree of apprehension, and when we are unable to account for the cause of the dyspnea, a bronchoscopic examination should be made. . . . In tracheobronchial diphtheria the process is a mechanical obstruction to respiration, plus carbonic acid poisoning and diphtheritic toxemia."

Lynah had seen several cases of tracheal diphtheria in private practice where the disease was early recognized. All these patients recovered after a dose of 10,000 units of antitoxin subcutaneously; there were parietal casts in all, but intubation was not necessary. "There is little or no prostration early in the disease, and postdiphtheritic paralysis is never seen, unless the case is of long standing and the pharynx and tonsils have become involved. Even enormous dosage of antitoxin and mechanical removal of the membrane in the case of long standing is futile, for these patients invariably succumb."

Lynah found it comparatively easy to remove the membrane in early cases, making use of a vacuum tube. The trachea and the bronchial system were then swabbed through the bronchoscope with antitoxin to prevent reformation of the membrane, and long

tracheobronchial tubes were introduced to prevent stenosis in case the membrane should reform and fill with blood clot.

During the ten years which elapsed after Lynah made his publication a number of single cases of diphtheria of trachea or bronchi were reported,[3 4 5 6 7 8 9 10] but the clinical picture described was practically identical, and all the comments were merely "rehashed" from Lynah, many being transcribed verbatim. Carpenter,[10] of South Carolina, saw, in consultation with a country practitioner, a five-year-old child, who had been ill for five days with what was supposed to be the aftermath of influenza. When seen by Carpenter the fauces, larynx, trachea and bronchi were covered with a diphtheritic membrane, although the practitioner assured him that there had been no signs of any membrane at his last visit to the patient only a few hours before the consultation was held. Carpenter passed a bronchoscope and extracted membranous casts from far down the bronchi and the trachea, which were necrotic, showing that the casts had been in existence for several days at least. The dyspnea was relieved, but the child died of diphtheritic toxemia.

A cursory examination of reports of diphtheria "carriers," which are extremely numerous in the literature, has not shown anywhere that the organisms were confined to the trachea throughout, as in our case under consideration.

Case Report.—A housewife, aged 67 years, first consulted us September 29, 1928. She gave a history of having had influenza 10 years previously and general poor health since, but at no time was she confined to bed. She had no cough except occasionally when a membrane was coughed up without difficulty. This had been going on for several months. Usually this membrane was a very small piece, though occasionally it was a piece as large as the little finger nail. Sometimes no membrane was coughed up for a period of a month, and then again pieces were coughed up within a few days or a week of each other, and accompanied by little or no mucus. During the interval there was no cough, no phlegm or mucus in the throat, and no soreness or pain in either throat or chest. So far as the patient knew there had been no fever.

Upon examination the nose and throat appeared essentially normal, and a very careful laryngoscopic examination disclosed

no abnormality in the larynx or trachea. As the patient appeared to be in fairly good health for a woman of her age, and was doing her own housework, she was advised to save some of the membrane coughed up and bring it for examination. Within a few days a small piece of dirty white membrane was brought in and submitted to a pathologist, who reported that both smear and culture showed diphtheria bacilli. The patient returned a few days later complaining of some discomfort in the windpipe, with a feeling of obstruction and slight disturbances in breathing. With the laryngoscopic mirror some of the membrane could be seen immediately beneath the vocal cords, and during the examination this membrane was coughed up and proved to be about the size of a ten-cent piece. This specimen was submitted to the pathologist, who again reported that in both smear and culture diphtheria bacilli were found. The patient had no fever and no increase in pulse rate. Chest examination was negative except for a few rales. Nose and throat were absolutely clear.

Forty thousand units of antitoxin were administered, without incident, and a few days later small pieces of membrane and some thick mucus were coughed up, and the pathologist reported that they gave an almost pure culture of diphtheria bacilli. Again 40,000 units of antitoxin was administered, but this had no particular effect. A week or ten days later more diphtheritic membrane was coughed up and again the pathologist reported the presence of diphtheria bacilli. During this treatment the patient had been feeling very well, was ambulatory, and exhibited no rise of temperature or pulse rate.

Upon the suggestion of the pathologist, autogenous vaccine treatment was started. The vaccine treatment was begun on October 15th, a dose just short of reactional being given every five days and the doses gradually increased. The vaccine treatment was continued with only short intermissions until August, 1929, or for a period of approximately ten months, and during that time membrane was coughed up at least two to six times per month, and in every instance the pathologist reported almost pure diphtheria bacilli. As a check, specimens were submitted to a second pathologist, who likewise reported the infection as diph-

theritic. Once during that period, or on November 5, 1928, the membrane assumed the form of a cast of the trachea and was about three-quarters of an inch in length. Five days later, or on November 10th, another smaller cast was coughed up. On August 27, 1929, the family physician decided to try toxin-antitoxin, and administered the regular course of toxin-antitoxin injections. This treatment seemed to be ineffective, so she returned to vaccine treatment, which was continued until November 23, 1929, when it was abandoned. Aside from the antitoxin, toxin-antitoxin and vaccine treatment, the patient had no other treatment except inhalations of compound tincture of benzoin vapor in connection with the inhalation of oily sprays.

The patient continues in fairly good general health, doing her own work, has no fever and has no cough except when cough is required to expel some of the membrane which still accumulates and is expelled on an average of two or three times a month. At no time since first seeing the patient, or until January 1, 1930, over a period of fifteen months, has there been any membrane above the larynx, nor could we procure diphtheria bacilli from the smears or cultures from tissues above the larynx.

A bronchoscopic examination never was made, as the patient objected to that procedure, though we are of the opinion that local treatment through the bronchoscope offers the best chance of relieving the condition and we would have employed the method had the patient consented.

As already stated, the case is unique and unusual, if we may judge by the literature of the last fifteen years, both in this country and abroad, which has been searched very carefully.

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*This reference is wrongly given in the Surgeon-General's Index as *Trousseau*, with the volume number as LII. This mistake was copied by Boebinger in his bibliography.

LXXIV.

THROMBOPHLEBITIS FOLLOWING A CASE OF CHRONIC OTITIS MEDIA.

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ST. LOUIS.

Patient's history: A female child, 10½ years of age, was brought to the St. Louis Children's Hospital in a semiconscious condition November 22, 1929. The patient's history as outlined was given by the mother.

The girl was the fifth child in a family of ten. Her early history was that of a normal breast fed child. No cod liver oil or orange juice in infancy or childhood. She had measles at two years with no complications; chickenpox at five. Before she was two years of age she began to complain of intermittent earache. After a severe "spell" of earache and crying, the right ear drum ruptured spontaneously and began to discharge. The child contracted cold easily and with each cold the ear discharged profusely. At all times this discharge had an exceedingly foul odor. As the child became older her hearing periodically improved. In the third grade, however, when the child was eight, her hearing was very defective and she had great difficulty in her school work. Because of the very poor condition of the child upon admission to our hospital we were unable to make hearing tests. From the mother we have a definite statement that the patient could always hear children better than she could hear adults. We have also carefully found out that there is no familial deafness.

Vertigo began with the first ear trouble and continued until her death. These dizzy spells were always accompanied by vomiting. They occurred on an average of every six weeks and were always diagnosed by the family physician as bilious attacks. For this the child was given calomel and salts, and usually, after a few days in bed, she would return to school or begin to play.

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The last illness began November 9, 1929, with the following symptoms: At supper she refused to eat. A short time later she had a severe chill. The mother gave her some calomel, but this time her condition became progressively worse. On November 13th, she suffered from severe dizziness. While up, the child showed a tendency to fall forward, being unable to balance herself without support. Severe vomiting occurred both when the patient was up and when she was lying down. On November 16th, she had a second violent chill. She became irrational and talked incoherently. The mother feared the child would go into a spasm and become unconscious.

A second doctor was called, who did not know what was the matter with the patient. The same day the child had a slight facial twitching on the left side. The mother observed practically total deafness at this time, making it necessary for her to shout loudly to make the child understand. On November 19th, she was brought to a hospital near their home, to a specialist. At that time she had developed a hacking cough.

Here she was given heat and light treatment. Two incisions were made through the right ear drum, one in the morning and the second in the evening (November 19th). No discharge after opening the middle ear. The following day a scant bloody secretion was washed from the right canal. A lumbar puncture, performed November 19th, was negative.

A brief resumé of her history in the first hospital reveals the following:

Urinalysis made November 19th, albumin a trace; a few R. B. C. and W. B. C.

No other laboratory report.

The X-ray report was as follows: "There is a definite veiling over the right mastoid, also a destruction of the cells; there is, no doubt, pus present."

Temperature chart:

November 19th—5 p. m., 104.4° F.; 10 p. m., 102.8° F.
November 20th—8 a. m., 99° F.; 5 p. m., 104° F.
November 21st—8 a. m., 99° F.; 5 p. m., 105° F.
November 22nd—8 a. m., 99° F.; 5 p. m., 104° F.

Treatment.—November 19th. Morning, right ear drum incised. Evening, operation repeated. No discharge from the ear. The ears were irrigated. Drops instilled into the nose. Light treatments were given. Lumbar puncture performed; negative.

The child was transferred to the St. Louis Children's Hospital in a semiconscious condition, at 9 p. m. November 22nd, very septic, slightly icteric, skin dry.

Despite an illness of two weeks with high temperature, chills, vertigo and vomiting, she at times rallied and asked to be taken home. In fact, before her transfer she had read mail from her school chums. (We consider this important in view of our post-mortem findings.) The child was very restless.

Lumbar puncture was immediately performed; the spinal manometer test gave no information.

Examination of the right ear drum showed a dull, retracted, gray membrana tympani with a slight sagging in the posterior superior quadrant; two recently closed incisions, one near the posterior margin of the annulus tympanicus and the other just posterior to the handle of the malleus.

Nose and throat essentially negative.

Eyes, slight exophthalmus of right eye. Pupils equal, react to light and accommodation. Pupils dilated with homatropin. Show bilateral choked disc. No tenderness over mastoid or along the jugular.

Laboratory findings on admission to Children's Hospital: White blood corpuscles, 16,500; polymorphonuclears, 70 per cent; lymphocytes, 10 per cent; mononuclears, 20 per cent; red blood corpuscles, 4,050,000; spinal fluid, 30 cells.

On admission to the St. Louis Children's Hospital we felt that the condition of the child was so poor that immediate operation would prove fatal. For that reason, after consulting with the pediatricians, it was decided that the best chance for the child was to give her Hartman's solution and a blood transfusion. The following chart gives the details of our method of procedure:

Preparation for Operation.—November 23rd, 1 a. m. 800 cc. of Hartman's combined solution, given subcutaneously.

9 a. m. 225 cc. of father's blood given intravenously.

2 p. m. 400 cc. of Hartman's combined solution, repeated. Temperature range at that time, 40 1/5 C.

After Hartman's solution and a blood transfusion were given, another consultation with the pediatrics staff was held, and it was the concensus of opinion that this was the best time to operate. Everyone realized that the only hope for the child was a ligation of the jugular and opening of the mastoid.

Operative Notes.—Time, 3:25 p. m. Anesthetic administered.

Usual preparation of neck and mastoid for right internal jugular ligation and mastoidectomy. The jugular was exposed for about 1½ inches, in the lower portion of the anterior triangle, by an incision passing through the anterior border of the sterno-cleido-mastoid muscle. The internal jugular vein was exposed and tied with two 20-day chromic catgut ligatures. The jugular appeared normal in this region. A simple mastoidectomy was then done. There was extensive sclerosis of the mastoid cortex and the cells of the mastoid in the region of the antrum. Over the lateral sinus the inner table of bone was black and necrotic. The cells of the mastoid antrum were thoroughly cleaned out and the antrum widely exposed. The dura over the region of the temporosphenoidal lobe was uncovered and found to be normal. The necrotic bone over the lateral sinus was removed for about 1½ inches backward from the knee and downwards toward the jugular bulb. Behind and below the sinus there was a well organized infected blood clot. The wall of the sinus was grayish-white and necrotic, and on opening the lumen was found to be filled with a partially broken down blood clot. This was removed until bleeding began posteriorly. The sinus was packed posteriorly with iodoform gauze. The same procedure was followed in opening the sinus below, toward the jugular bulb.

The patient collapsed at the end of the operation and died shortly after being taken from the table to the room.

Blood pressure rapidly fell. Pulse and respiration likewise weakened. Patient expired at 4:35 p. m.

ABSTRACT OF AUTOPSY PROTOCOL.

Anatomical Diagnosis.—Thrombophlebitis, septic infarction of lung, blood in kidney tubules. (The detailed description of body organs is omitted, as nothing particularly striking is shown.)

"Brain.—When the skull cap is removed no change is seen in the dural covering of the brain. After the dura is removed the brain substance is revealed and found to be covered with a thin tenacious material which is light brown in color and which covers the brain in so fine a layer as to be almost invisible. When the brain is removed there is found an accumulation of fluid in the posterior fossa which is also light brown in color and turbid in character. There is, however, no macroscopic exudate on the basilar portion of the brain, and no other changes are seen on its surface. Section of the organ reveals only a few dilated blood vessels.

Hypophysis.—No gross change is seen in the hypophysis.

Sinuses (Venous).—The sinuses are investigated to ascertain whether thrombi are present or not. The superior sagittal sinus is found to be free from any such change, as are the transverse and petrosal sinuses on the left side. However, a thrombus is seen to extend in the transverse (lateral) sinus on the right side from its beginning at the junction with the sagittal sinus throughout almost its entire course. This thrombus does not, however, extend into either the superior or inferior petrosal sinuses on this side, and the basilar plexus and cavernous sinus are also uninvolved. The thrombus which lies in the right transverse (lateral) sinus is quite obviously infected, and purulent material may be seen in its substance. It does not, however, extend through the dura, and the brain substance beneath it is apparently free from change.

Temporal Bones.—The petrous portions of the temporal bones are removed from both sides. The one on the right shows the evidences of the operative procedures mentioned above, but there is no evidence (grossly) of direct spread of infection from this portion of the bone into the transverse (lateral) sinus. The opposite bone shows no change from the outside. These are delivered in toto to the ear, nose and throat department.

Nasal Cavity and Accessory Air Sinuses.—A section of the sphenoid bone with the air sinuses, part of the vomer and nasal septum is removed en bloc and delivered to the ear, nose and throat department. From the superficial gross inspections of the

material allowed at autopsy no change is seen. (Microscopic sections practically negative.)

Microscopic. — (Other organs essentially negative except lungs.)

Brain.—Brain stem, no changes except a few dilated blood vessels at the base. Another section is taken from the parietal lobe on the side operated upon. This section also shows some dilated blood vessels on its meningeal surface, but in addition there is some hemorrhage and exudation of cells along the meningeal surface, at a point where the organ was in contact with the exposed dural surface. There is no extension of the process to cover the surface of the brain.

Hypophysis.—The cells of the anterior lobe of the hypophysis are swollen and stain darkly, but no other changes are noted in this organ.

Lateral Sinus.—A section of the lateral sinus on the right side is made at the site of gross thrombosis. Section shows the walls of this structure to be thickened and invaded by inflammatory cells. In some places this is in direct extension with the infected area which is seen on the outside of the dural coat, but the entire wall is involved. Many of these cells which have infiltrated the sinus wall are polymorphonuclears, and they group themselves about the blood vessels in especially large numbers. In the lumen of the sinus is a mass of blood clot, polymorphonuclear cells, necrotic material and bacteria. This serves to almost occlude the portion of sinus seen in section.

Dura.—A section of the dura which was exposed at operation shows an infected outer surface, which is covered with red blood cells and a few polymorphonuclear cells. Bacterial stains show some bacteria in these areas. The dura itself is infiltrated with polymorphonuclear cells which extend to the inner surface but do not break through it. Many dilated blood vessels may be seen in such areas.

Jugular Vein.—A section of the jugular vein on the right side shows no microscopic change. (Not stated where taken.)

Ear.—Section of the right ear shows that the drum is intact. In the middle ear cavity there is an accumulation of inflammatory cells, most of which are red blood cells and polymorphonuclears. This inflammatory change may be seen extending down the eustachian tube from the middle ear cavity. The submucous layer of the eustachian tube is quite thick and edematous. It is infiltrated with many polymorphonuclears and round cells, which extend throughout the portion seen in section. No other changes are noted in the ear.

Summary.—Autopsy reveals a middle ear infection, which has extended into the mastoid bone. An infectious thrombosis of the lateral sinus is also noted with the involvement of the sinus wall. Embolic phenomena are noted in the lungs, where septic infarcts with abscess formation are found. The blood in the tubules of the kidney may be a manifestation of an early acute nephritis. No organisms are stained in this latter organ."

POSTMORTEM BACTERIOLOGICAL FINDINGS.

Material Cultured.—1. Smear of meningeal pus. 2. Lateral sinus pus. 3. Heart blood culture. Results.—1. Smear showed gram positive staphylococcus. 2. Smear showed gram positive staphylococcus. 3. Heart blood positive for staphylococcus albus. White pigment producer.

Conclusion.—Staphylococcus albus. Sinus infection and septicemia.

MICROSCOPIC STUDY OF TEMPORAL BONES.

A careful study of serial sections of the temporal bones of this case reveals conditions which add materially to information regarding the course of the disease. The study also proves that the complete story so necessary to the otologist can by no means be gained from the diagnosis of a single section. It is necessary to have serial sections to secure all information. This may be seen by making a comparison of the brief note on one ear section, included in the protocol, as compared with the photographs and descriptions we present below. From each series made in the otologic laboratory one section through a supposedly sig-

nificant area is routinely sent to the pathological department. The rest of the series is examined in detail in the otological laboratory.

The sections were prepared according to the routine celloidin technic. The right bone was sectioned horizontally, the left vertically, each at 20 micra.

The two series present a picture quite in keeping with the history of the case. The sections show an old chronic infection in both ears, that on the left being the older. On this side no active suppuration in the middle ear is seen but marked bone changes are present. Active suppuration is present on the right, however.

The acute condition on the right, which was causing the trouble at the time of death, was further aggravated by the presence of a large cholesteatomatous mass, the presence of which was not suspected before death. (It is to be remembered that "cholesteatoma" in the middle ear does not have the same connotation as "cholesteatoma" in general pathology. Cholesteatoma of the brain, for example, is quite different in origin. In the brain, cholesteatoma originates as a misplacement of ectodermic germinal cells, which in later life are suddenly stimulated to growth. In cholesteatoma of the middle ear, however, the ectodermal misplacement can arise from a direct extension of the epidermis of the external auditory canal into the middle ear via a perforated or ruptured drum membrane.) We insert a few lines on the subject of cholesteatoma from Politzer, "Diseases of the Ear," pp. 454-455:

"Cholesteatoma in the ear has been long known to pathological anatomists, as is seen from the writings of Cruveilhier, Rokitansky, Virchow and others. There is still, however, a great diversity of opinion in regard to the origin of these accumulations of epidermis. Virchow, Mikulicz and Kuster explain the occurrence of cholesteatoma in the temporal bone as a heteroplastic formation; v. Troltsch regards it as a retention tumor; Wendt, as the product of a desquamative inflammation of the mucous membrane of the middle ear; while Bezold and Habermann consider it the result of the inward growth of the epidermis of the external ear through the perforation of the membrane into the tympanic cavity. Leutert distinguishes a real tumefaction and

cholesteatoma, brought about by the implantation of the remnants of the tympanic membrane, which are covered with epidermis . . . . Clinical and pathological observations have shown beyond a doubt that the formation of cholesteatomata of the ear is secondary in the great majority of cases."

The cholesteatomatous mass lay in the middle ear cavity in such a position as to block the escape of pus from the two perforations made in the drum membrane (photo II, section 347, and photo VII, section 496). It extended high into the antrum —in fact, to the level of the arch of the superior semicircular canal. In passing through the attic it so blocked in the pus in that region that the mucous membrane enveloping the ossicles was invaded, with the concomitant result that severe joint infection has occurred in the malleoincudal joint. The periosteum is gone and the osseous tissue itself has been attacked.

Infected thrombi in the venous plexus surrounding the internal carotid artery are seen in each ear. (It will be recalled that this plexus communicates with the sinus cavernosus above.) On the right side there are indications that the greater part of this thrombus has been dislodged either at autopsy or earlier (probably when the organized thrombus of the lateral sinus was removed at operation). The impress is still obvious. (Photo I, section 347.) On the left side the thrombus is highly organized. (Section 110, etc.).

The niche of the round window in each ear is almost entirely filled in with dense fibrous connective tissue. That on the left is closely adherent to the secondary tympanic membrane, giving it the effect of a greatly thickened membrane (photo X, section 747). On the right the fibrous tissue has not filled the niche so completely. (Photo VII, section 496.) This latter condition is probably merely a matter of more recent invasion.

For the sake of clarity an outline of the salient findings of each ear will be given in the chart below:

MICROSCOPIC FINDINGS.

Right Ear.	Anatomical Region.	Left Ear.
	TYMPANIC CAVITY.	
The tympanic cavity contains: 1. Cholesteatomatous mass walled in by stratified squamous epithelium. The mass extends from the level of the arch of the superior canal (Sec. 80) to the level of the oval window (Sec. 365). The mass measures approximately 5.7 mm. x 5 mm. x 2 mm. (See Photo I, Sec. 347.) 2. Débris and necrotic material not walled in by epithelium, but blocking the lower incision at the level of the round window. (Photo VII, Sec. 496.) 3. Fragments of bone (Photo II). 4. Free blood and pus (Photo I). 5. Dense fibrous connective tissue in the niche of oval window (Photo I) and in that of round window (Photo VII). This tissue we believe to be scar tissue. Neither this nor the edematous mucosa leading toward the eustachian tube can be mesenchyme remnants such as described by Dr. Crowe (Archives of Otolaryngology, August, 1930). We have compared the tissue microscopically with mesenchyme of a six months foetus and find this to be of quite different character. A nuclear count shows the cells in the fibrosed submucosa to be much more numerous per given area than those in the embryonic tissue. The matrix of the embryo is extremely delicate and stains bluish while that in the fibrosed region is coarse and dense and has a greater affinity for the eosin. The nuclei of the embryonic tissue show much more abundant exoplasm (giving the characteristic star shape) than do those of the case in question. Rounded spaces within the matrix evidence edema while the ground substance of the embryonic tissue is evenly spread.		1. Negative except for hyperostoses protruding into it, notably in the region of oval window. (Further discussed under bone.) Photo XII. 2. Dense fibrous connective tissue of very old type in niche of round window.

MICROSCOPIC FINDINGS (CONTINUED).

RIGHT EAR.	ANATOMICAL REGION.	LEFT EAR.

MUCOUS MEMBRANE—SUBMUCOSA.

1. Highly edematous and containing many cystic spaces. Evenly infiltrated with small mononuclears just below the epithelium. In places these monos center in patches. Extravasated R. B. C.'s are present as are large mononuclears, a few polymorphonuclears and in certain areas, plasma cells (especially in the dense tissue binding in the footplate of stapes). (Sec. 348).

2. Cystic spaces contain a serous exudate and numerous very large cells (mostly necrotic) containing one or more nuclei. These possess pseudopodia. (Sec. 360-380.)

1. Submucosa densely fibrous and necrotic.

EUSTACHIAN TUBE.

1. Filled with free blood and pus. (Photo I, Sec. 347).

2. Simple columnar epithelium with cilia visible down to end of bony and cartilaginous wall.

3. Median wall—submucosa highly edematous, infiltrated with mononuclears and polymorphonuclears. Cystic condition present.

1. Lumen clear.

2. Finger-like projections of the mucous membrane, resembling villi, protrude into the tube. (Sec. 370.)

3. Submucosa not greatly thickened but containing many blood vessels and much dense fibrous tissue. (Sec. 492B.)

4. Near tympanic cavity, processes of bone covered with epithelium project into the lumen in places. (Sec. 906.)

OSSICLES.

1. Joint infection in malleo-incudal joint especially on the medial side. (Photo IV, Sec. 203.) For detail see Photo V and VI. Here the periosteum on the one side is completely gone leaving a ragged edge on each ossicle. Osteomyelitis is present in the incus. In Photo V the joint capsule proper is shown still intact but the mucous membrane is breaking down.

2. Incus is dislodged from the incudostapedial joint.

3. Drum membrane is adherent to stapes. (Photo III, Sec. 410).

1. Photo XI, Sec. 854 shows a practically normal malleo-incudal joint. However, this joint was attacked in one place also. (Sec. 906.)

2. Sec. 854 shows the malleus displaced and in almost complete contact with a spicule of bone projecting from the epitympanic wall, an example of the many marked bone-changes on this side.

3. Foot plate of stapes evidently lacked calcium as it presents an undulating appearance, while the embedding celloidin is here without wrinkle or fold. (Sec. 734.)

MICROSCOPIC FINDINGS (CONTINUED).

RIGHT EAR.	ANATOMICAL REGION.	LEFT EAR.
	OSSICLES—CONTINUED.	
4. Stapes and stapedial muscle are completely bound down by dense connective tissue. (Photo I.) Photo VIII shows the apex of the pyramid on the posterior wall of the tympanic cavity from which the stapedius muscle emerges and goes to the capitulum of the stapes. As stated the muscle is completely surrounded by connective tissue.		4. Projecting toward the stapes in the niche of the oval window are two exostoses suggesting those seen in otosclerotic cases. (Further discussed under bone.)
	TYMPANIC MEMBRANE.	
1. Thickness: Varies from .8 mm. to .175 (Sec. 347-203). This shows that in places it is 8 or 10x normal. Stratified squamous epithelium on the middle ear side. (Sec. 300.) Heavily infiltrated with polymorphonuclears and extravasated R. B. C.'s. Vessels much congested. 2. Tympanic membrane adherent to head of stapes. (Photo III, Sec. 410.) Photo VII, Sec. 496 shows the lower perforation healed on the posterior edge but not on the anterior edge. The perforation is more or less occluded by necrotic material which is here *not* surrounded by stratified squamous epithelium. Photo IX, Sec. 422 shows a region of the tympanic membrane intact throughout, where no perforations are indicated, nor any cholesteatomatous mass with misplaced squamous epithelium. A diagnosis from such a region alone would give an erroneous idea of the pathological condition.		1. Thickness: .025 mm. Not intact. Atrophied in areas.
	SECONDARY TYMPANIC MEMBRANE.	
1. Torn (probably artefact). Fibrous tissue filling in the niche on this side does not quite reach to the secondary tympanic membrane as on the left. (Photo VII, Sec. 496.)		1. Greatly thickened and niche practically filled in with very old dense scar tissue. (Photo X, Sec. 747.)

MICROSCOPIC FINDINGS (CONTINUED).

RIGHT EAR.	ANATOMICAL REGION.	LEFT EAR.
	EXTERNAL AUDITORY CANAL.	
1. Desquamation products present. Thickened subcutaneous layer on median wall.		1. Subcutaneous vessels enlarged and multiplied particularly along the inferior external wall. The vessel involved is undoubtedly the deep auricular, a branch of the internal maxillary (Politzer). The epidermis is keratinized and sloughed off. (Photo XI, Sec. 854.)
	NERVES—CHORDA TYMPANI.	
1. Photo I, Sec. 347 shows an unusual longitudinal cut of the chorda tympani as it courses toward the malleus.		1. Negative
	FACIAL NERVE.	
1. Diamond shaped spaces suggesting cholesterin crystal imprints but no crystals were demonstrated; so this may be merely shrinkage after extreme edema. 2. Mural thrombus in stylomastoid vein accompanying facial nerve. (Sec. 430.) 3. Evidence of pressure atrophy of nerve plexus surrounding internal carotid artery. This condition was due to the existence of a thrombus in the venous plexus in this region. (Discussed under blood vessels.)		1. As on right.
(Eighth nerve discussed under "Inner Ear.")		
	BLOOD VESSELS.	
1. Fragment of bone in carotid artery, also pus, Sec. 420. This small spicule of bone was probably forced into the carotid artery at the time of the autopsy. (Small infected thrombus.) 2. Photo I, Sec. 347 shows the impress of a thrombus in the venous		1. Organized thrombus in venous plexus surrounding internal carotid artery. (Sec 110.) 2. Excessive vascularization and congestion of involved vessels in subcutaneous area of external auditory canal just outside of tympanic membrane.

MICROSCOPIC FINDINGS (CONTINUED).

RIGHT EAR.	ANATOMICAL REGION.	LEFT EAR.
	BLOOD VESSELS—CONTINUED.	
plexus in the internal carotid canal. This has been dislodged either at autopsy or before death (probably when the thrombus was removed from the lateral sinus at operation.) 3. Vessels accompanying facial nerve appear thick walled. There is a mural thrombus in the stylomastoid vein in the facial canal and numerous polys in the stylomastoid artery. 4. Congestion of cochlear artery.		3. Atrophy and complete disappearance of cochlear artery in upper part of axis.
	BONE.	
1. Late closure of fissures. 2. Excessive amount of fat in marrow spaces in apex of temporal bone. 3. Form of bony capsule (basal cochlear turn) distorted. 4. Canal walls irregular. 5. Irregular walls of endolymphatic duct. (Vestibular aqueduct.) 6. Osteomyelitis in incus, as noted above.		1. The most striking pathological features of this ear are found in the bony changes. Marked invasion of bony areas by dense fibrous connective tissue, along the middle ear wall. Exostoses exist concomitant with resorption of bone by the invading connective tissue. These malformations resemble in contour the structures studied by one of the authors in a case of bilateral otosclerosis in the Johns Hopkins Otological Laboratory. In contrast to the typical picture of otosclerosis, however, there is no blue staining center, "Herde", of new-formed bone sending out radiating processes into the old bone. Here we have only an extreme vascularization in the center of each exostosis. The vessel involved in the lower exostosis, at least, seems to be that which supplies the superior wall of the promontory, that is the superior tympanic artery (a branch of the middle meningeal). 2. Chronic passive congestion is present in these vessels. 3. Intrachondral bone (Bast) almost totally absent.

MICROSCOPIC FINDINGS (CONTINUED).

RIGHT EAR.	ANATOMICAL REGION.	LEFT EAR.
	MUSCLES.	
1. Stapedius muscle completely embedded in connective tissue. 2. Fatty atrophy of tensor tympani.		
	INNER EAR—(A) COCHLEAR NERVE.	
1. Atrophy of spiral ganglion cells, especially basal turn. (Sec. 350.)		1. Spiral ganglion atrophied. (Sec. 492.) 2. Complete atrophy of organ of Corti in upper turns of cochlea. Remnant in basal.
	(B) VESTIBULAR NERVE.	
1. Vestibular ganglion normal.		1. Vestibular ganglion fairly normal. (Sec. 492.)
	(C) ENDOLYMPHATIC CAVITIES.	
1. Serous labyrinthitis present. 2. Practically complete atrophy of crista in posterior ampulla. There is dense labyrinthitis present. 3. Endolymphatic duct with a very heavy wall. No evidence of purulent infection.		1. Perilymphatic tissue atrophic. 2. Atrophy of maculae of saccule and utricle. (Sec. 764.) 3. No evidence of purulent infection.
	(D) SPIRAL LIGAMENT.	
1. Densely fibrous and atrophic with beginning atrophy of stria vascularis.		1. Atrophy more marked than on the right. Stria a mere pinkish band.
	(E) BLOOD VESSELS.	
	COCHLEAR A.	
1. Marked congestion.		1. Complete atrophy and disappearance of cochlear artery in apex of modiolus and in stria. 2. Walls extremely thick in the region of the internal auditory meatus.

CONCLUSION.

Right Ear.—The outstanding clinical lesson in this case is that the drum membrane picture is not always pathognomonic of the underlying microscopic and destructive picture. Four days before the child's death two incisions were made into the tympanic membrane to establish drainage. The appearance of the drum on admission was that of a dull, gray, retracted membrane, with only the slightest suggestion of sagging in the posterior superior quadrant. The severity of the clinical symptoms must always be the guiding point for early operative procedures. In this case there was an extensive effort on the part of nature to wall off the infection—e. g., cholesteatomatous material surrounded by stratified squamous epithelium, but no evidence of this presented itself either in the drum picture or in tenderness over the mastoid. Some time before the present illness the right tympanic membrane must have ruptured spontaneously and in the healing process epidermis of the external surface migrated into the middle ear cavity. Its presence there was probably followed by desquamation (a normal process for stratified squamous epithelium). But there was no easy exit for the desquamated material—hence the building up of the cholesteatomatous mass. It required the most intensive postmortem study of microscopic serial sections to demonstrate the degree of the underlying pathology. A study of photo IX, sec. 422, is practically negative. From our study it is obvious that a picture is only complete when the entire temporal bones are serially sectioned throughout.

Left Ear.—There was nothing in the clinical picture to draw our attention to the left ear. Simply a slight retraction of the drum membrane. It is highly probable that this ear gave the child difficulty in earlier life, but that the mother (a mother of ten children) either did not know it or had forgotten that the left also was at one time involved. This labyrinth must certainly have been practically dead. The delicate perilymphatic tissue, accurately described by Politzer as being highly vascular, is nothing but an atrophic, necrotic and very open meshwork.

To Dr. L. W. Dean, who established the otologic laboratory in Washington University, we feel highly indebted for his guid-

ance. We wish to thank Dr. H. W. Lyman for his valuable clinical direction, and also the departments of pediatrics and pathology.

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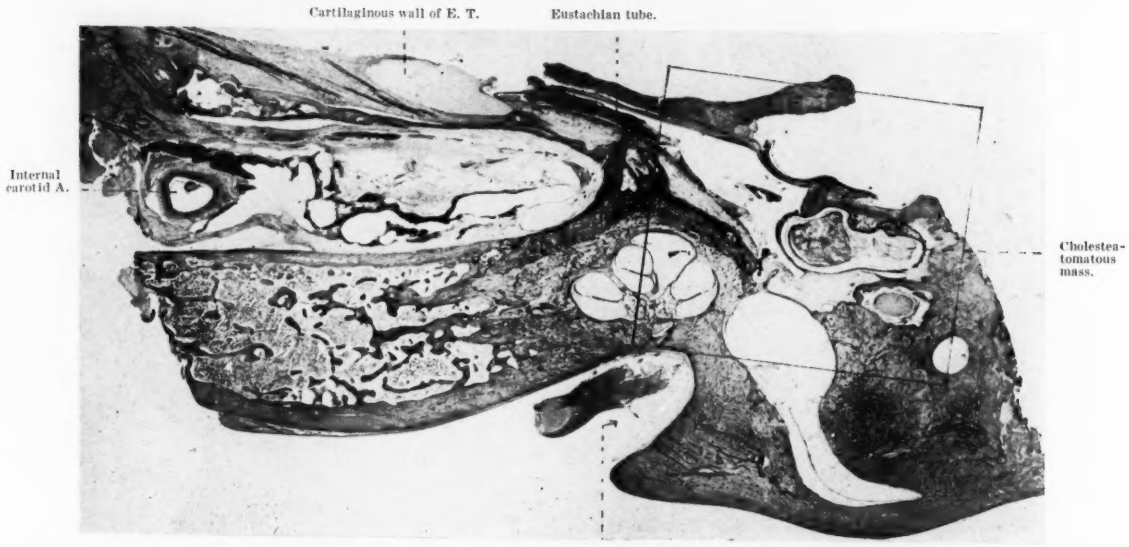

Photo 1. Sec. 347—Right. Shows foot-plate of stapes bound down with old scar tissue; cholesteatomatous mass in middle ear; a fragment of bone in middle ear cavity; blood and pus in eustachian tube; longitudinal section of chorda tympani above cholesteatomatous mass; carotid canal with internal carotid artery in x-section at extreme left; venous plexus showing partly dislodged thrombus.

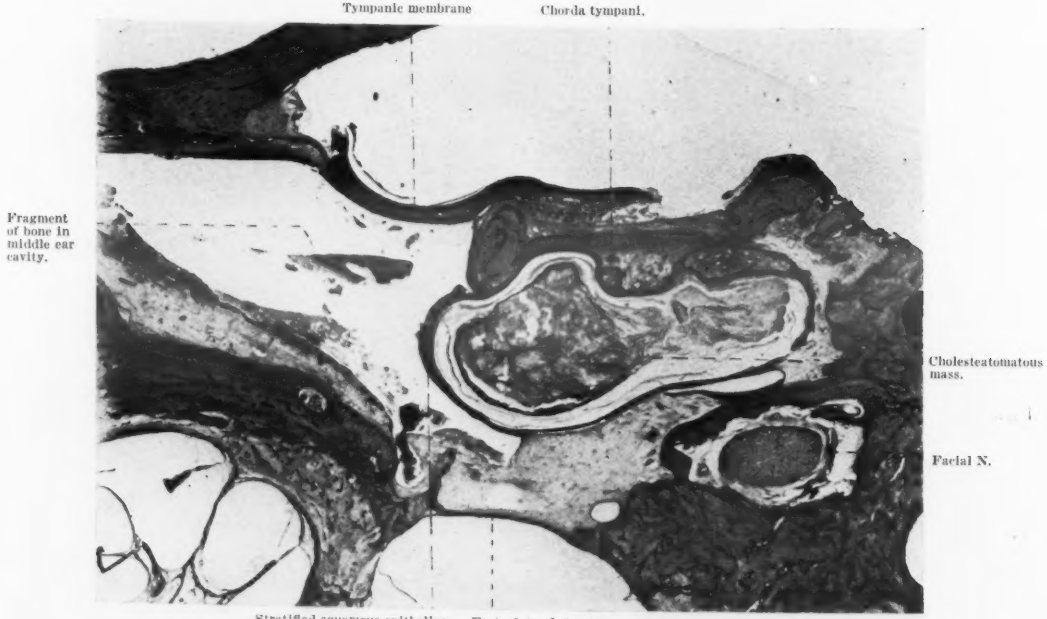

Photo II. Sec. 347—Right. Detail to show longitudinal cut of chorda tympani; foot-plate of stapes bound down by scar tissue; cholesteatomatous mass surrounded by stratified squamous epithelium; drum membrane perforated above chorda tympani.

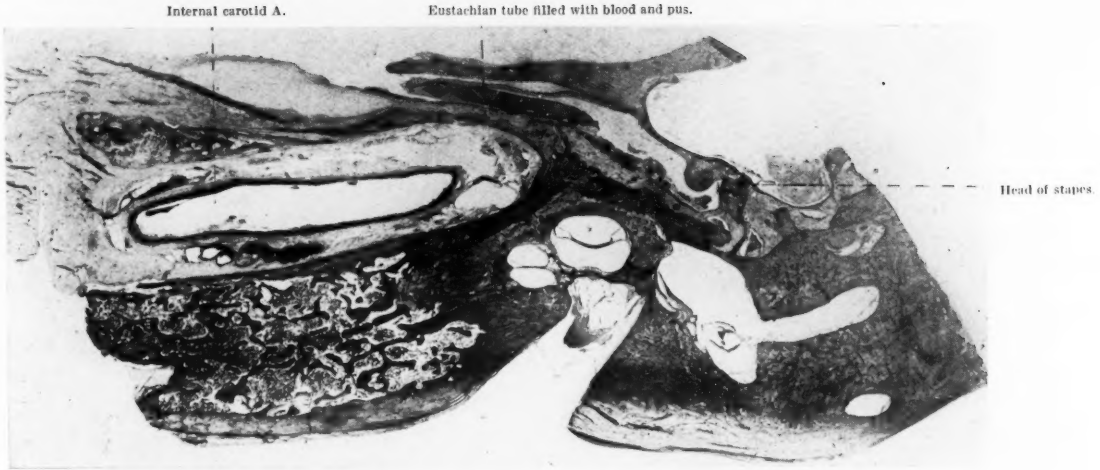

Photo III. Sec. 410—Right. Shows drum membrane bound to head of stapes and nitch of oval window filled with connective tissue; longitudinal cut of carotid artery in carotid canal.

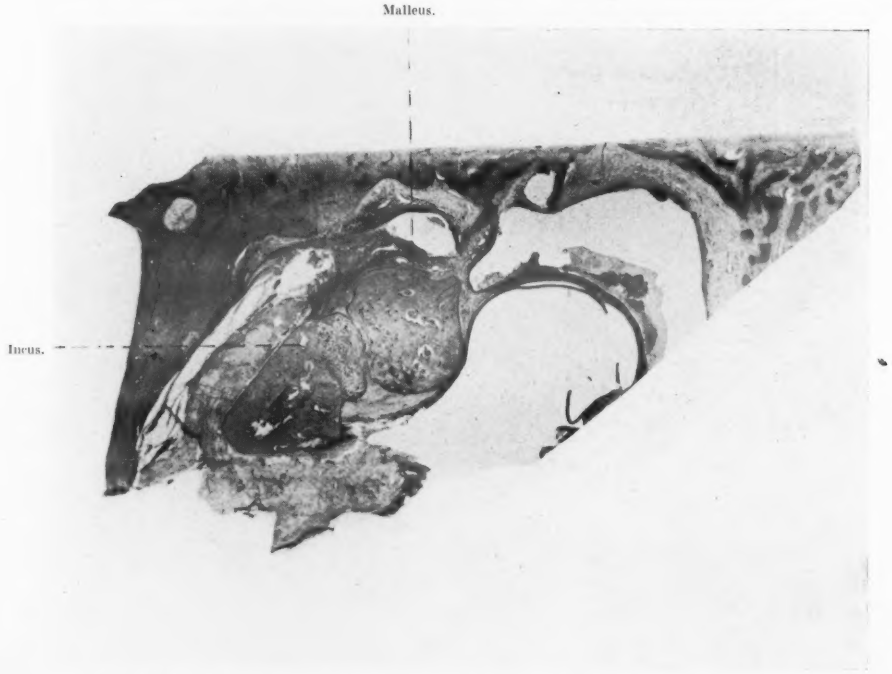

Photo IV. Sec. 203—Right. Shows infected malleoincudal joint, and cholesteatomatous mass.

Malleus.

Incus.

Photo V. Sec. 203—Right. To show more normal side of malleoincudal joint.

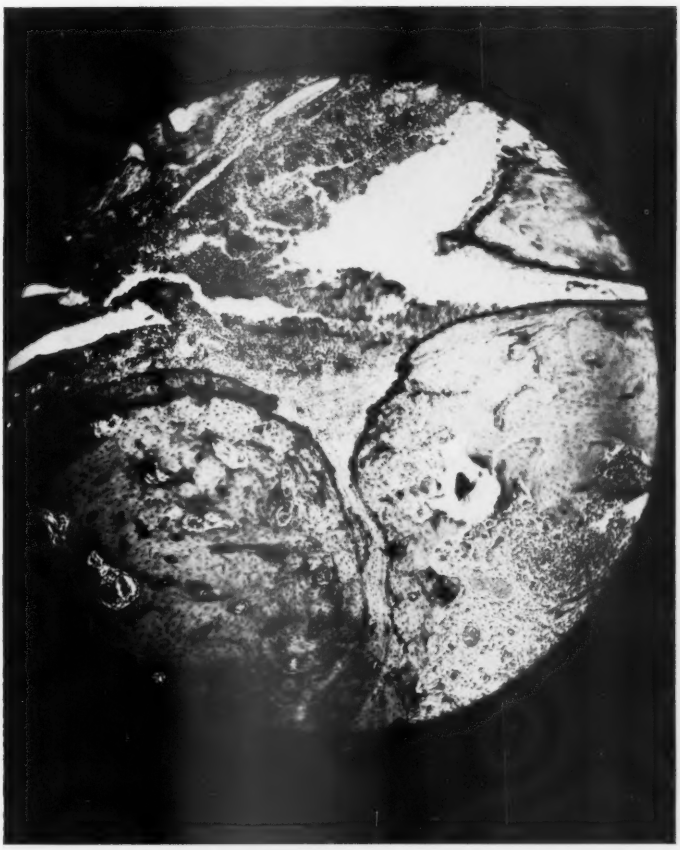

Photo VI. Sec. 203—Right. To show heavy infection of malleoincudal joint.

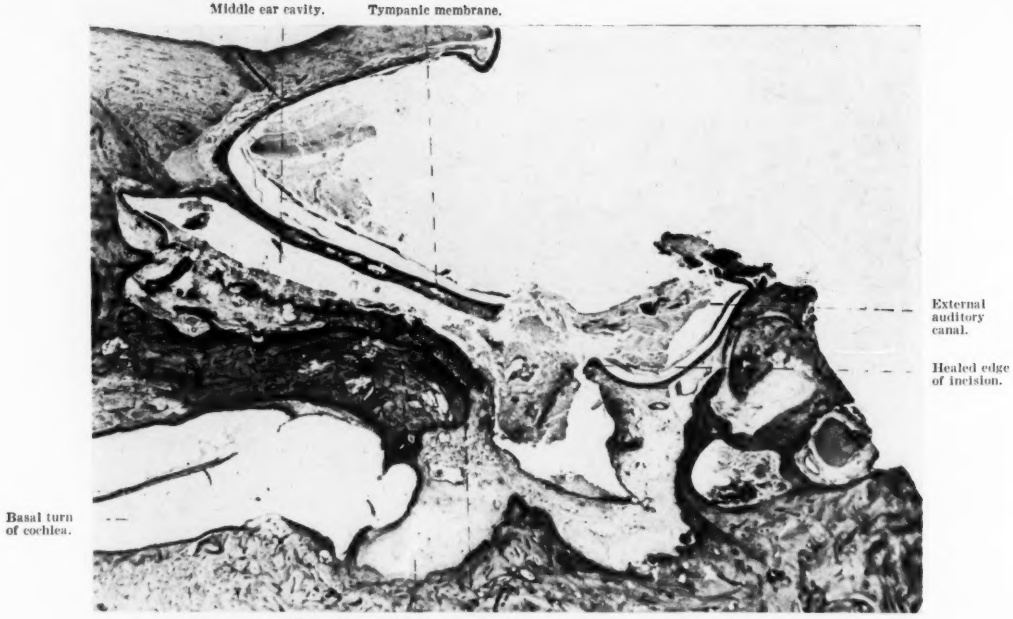

Photo VII. Sec. 496—Right. Horizontal section at the level of the round window and of the lower paracentesis, the posterior edge of which is healed. The aperture is more or less blocked with debris. The niche of the round window is almost completely filled in with dense fibrous connective tissue. The secondary tympanic membrane itself is torn. (Probably artefact.)

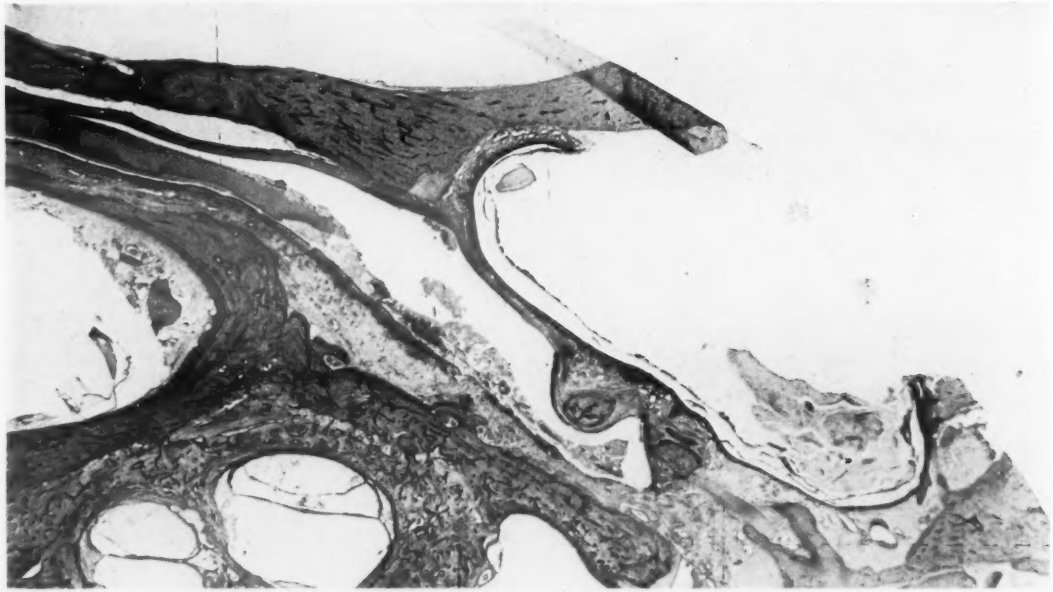

Photo VIII. Sec. 409—Right. Shows the pyramid from which the stapedial muscle emerges and extends to the head of the stapes. The muscle is completely embedded in dense fibrous connective tissue.

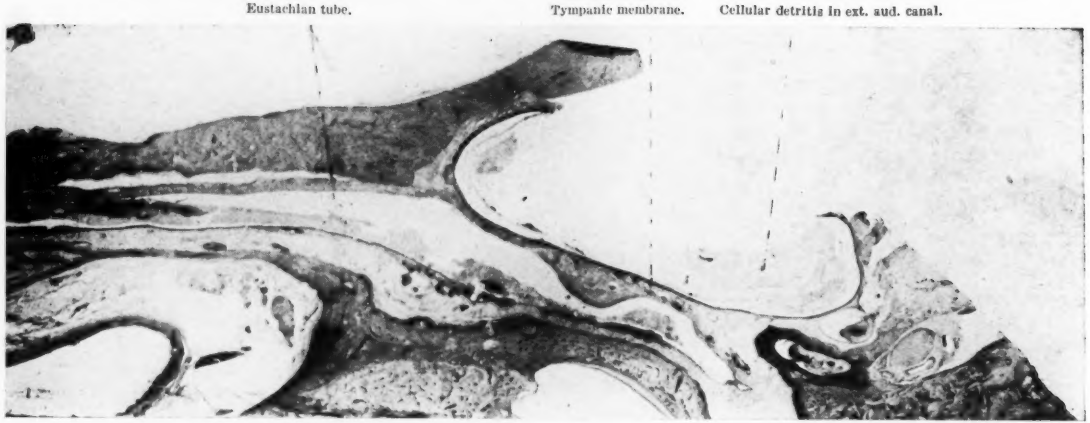

Photo IX. Sec. 422—Right. Shows tympanic membrane edematous but intact throughout, with no indication of perforation or of cholesteatoma. This region lies between the levels of the two perforations.

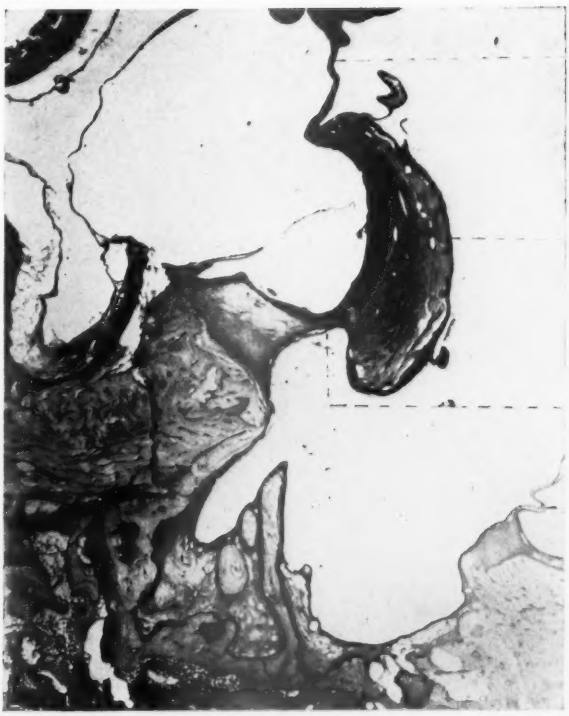

Photo X. Sec. 747—Left. Vertical section at the level of round and oval windows. The niche of round window contains old dense fibrous connective tissue. Note bend in stapedial foot-plate.

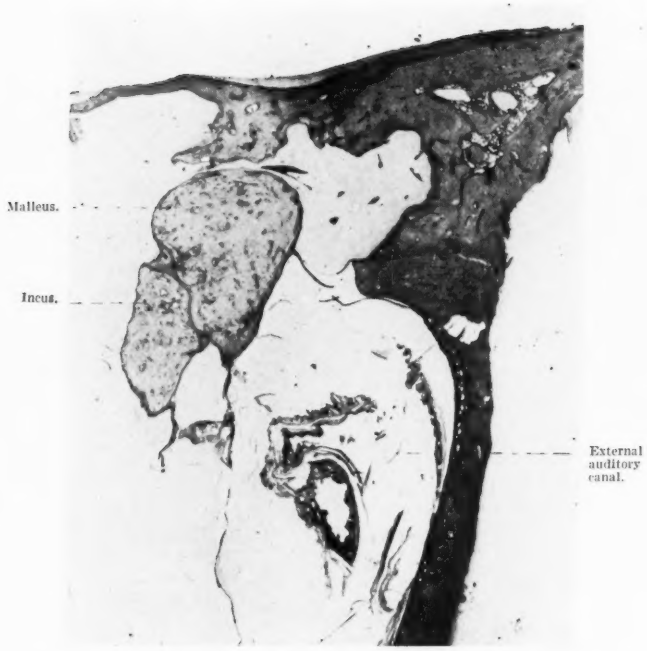

Photo XI. Sec. 854—Left. Fairly normal malleoincudal joint; malleus almost ankylosed to tympanic wall. Drum membrane displaced. Keratinized epidermis in external auditory meatus.

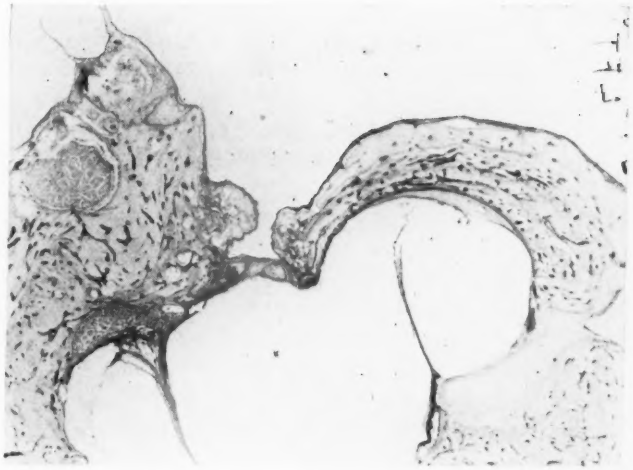

Photo XII. Sec. 680—Left. This section shows exostoses extending over oval window. The niche of the round window is also filled in with dense fibrous connective tissue.

LXXV.

RELIEF OF RIGHT-SIDED HEADACHE BY RESECTION OF LEFT SPHENOID (ANATOMIC VARIATION OF SPHENOID SINUSES): REPORT OF A CASE.

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ST. LOUIS.

The identity of nasal headache has been so well established that we often form an accurate opinion of the sinuses involved from the history alone. We expect a lesion of certain sinuses or groups to refer pain to a fairly constant surface area. A suppurative process will appear at usual intranasal landmarks. Simple X-ray study usually shows changes in density due to sinus disease and variations in structure. The development of lipiodol study enables even greater sinus detail to be made out. It is therefore most difficult to deviate from the habit of expecting the sinus lesion to be on the same side as the expression of pain. The rare occurrence of a sphenoid cell extending to the opposite side is not usually considered.

Onodi,[1] in showing the frequent influence of one sphenoid cell on disease of both optic nerves, collected a number of cadaver specimens of unusual morphologic arrangement of sinuses. He observed dehiscences in the septal wall of the sphenoid sinuses, and referred to similar cases in the work of Zuckerkandl. Hajek[2] described total absence of the septal wall of the sphenoid sinuses, and other cases of such marked asymmetry of the cells that the larger of the two extended through the entire coronal plane of both and behind the smaller. It is the latter variation that closest resembles the case presented herewith, diagnosed by means of radiopaques and proven by operation.

Sluder[3] encountered many cases of this type in his large clinical experience and felt that they could be diagnosed only when

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found at operation. He resorted to resection of the posterior cells on the opposite side when operation on the indicated side failed to relieve what was clearly posterior sinus disease.

The correct analysis of this case was made possible by the unilateral introduction of lipiodol into an unrecognized sphenoid cell by displacement with the head in the Proetz[4] position. Repeated surgical measures had been instituted on the posterior sinuses by various competent nasal surgeons, and had failed to locate the cell responsible or to relieve the headache, which was felt to be, by elimination of all other sources, nasal in origin.

Case report: Mrs. S., age 59, presented herself July 2, 1929, complaining of constant, daily, severe headache for several years, for which in the past two years she had undergone two or three nasal operations, with no relief. The pain was located always on the right side, in the right root of the nose, over the right eye, and in the right vertex and occiput, so severe at times that she became nauseated. She had much sneezing but no nasal obstruction.

The past history was entirely negative, with never any disabling illness until the present complaint began.

Objective examination of the nose was recorded as follows: "Total resection of all posterior cells, with the removal of middle turbinates; some webbing on the right side encloses recesses, from which is pouring yellow pus; the right sphenoid is open. On the left there is no webbing, and all cells are well open and clean." Diagnosis: Postoperative occlusion of the right sphenoid sinus, with scarring.

Unilateral X-ray study of the posterior sinus region was made, introducing lipiodol by displacement with the patient's head in the Proetz[4] position. It showed the left sphenoid cell to be present and intact and varying from the normal position. The lipiodol instillation was made first on the right and several days later on the left, making plates of the anteroposterior plane, lateral, and lateral with the head in the Proetz position.

Previous careful inspection of the nose seemed to show every possible posterior cell removed, and no ostium to indicate the presence of another cell. Measurement along Zuckerkandl's line from the inferior nasal spine to the face of the left sphenoid was 6.2 cm., and to the back wall of the open right sphenoid was 6.6 cm. This measurement bordered on normal for the back wall of either sphenoid cell and was again convincing evidence that all cells had been removed. As shown in Figs. 3 and 4, the lipiodol passed into a large left sphenoid cell, behind a very shallow right one. It extended across the midline, as shown in the figure and later proven by operation. The right cell was wide open and easy to identify, but there was no proof of the presence of the left until the lipiodol study was made.

Sluder,[3] in considering the occasional anomaly of one sphenoid cell extending into the other side, stated that he had no means

of identifying this anomaly, and bearing the variation in mind he operated upon both sides in some desperate cases where the result on the indicated side was a failure.

Fig. 1 is a study of the right side, plate of anteroposterior plane in upright position, shows the forward lying cell to extend into a pneumatized pterygoid process, identifying it as sphenoidal.

In Fig. 2 the right side of the nose is filled with lipiodol by displacement, with head in Proetz position, shows lipiodol settled into a shallow, forward lying sphenoid cell, with a large sphenoid cell space behind it.

Fig. 3 is a study of the left side. A posteroanterior film shows lipiodol extending across to the right side and defining the exact lateral extent of the left sphenoid cell.

In Fig. 4 the left side of the nose is filled with lipiodol by displacement. With the head in the Proetz position, it shows lipiodol admitted to a large sphenoid cell and kept from settling into another depression by what was later found to be a large polyp.

In Fig. 5 the probe is resting in the widely opened face of the right sphenoid. The curved cannula is in the unopened left sphenoid, which is filled with lipiodol. Information from the probes could give no hint that one cell extended across the midline, and no deduction could be made if all posterior cells were filled with lipiodol. The difference could only be made out by unilateral lipiodol study. The thickened cell mucosa is well shown against the sella. The concave deformity of the shadow forward and above were later found to be large polyps in the sphenoid.

Operation: Under local anesthesia, the face of the left sphenoid was readily broken down, disclosing a cell of enormous size, from which several lobes of a large polyp pushed outward the instant the opening was made. The polypoid mass actually seemed to be under pressure. It was removed, and a sound could be passed across the midline, confirming the extent of the sinus, as shown in the X-ray study.

The postoperative course was uneventful, the patient stating that the pain was remarkably relieved. After two and a half years there has never been another attack of the profound right-

sided pain with nausea. On several occasions she has had antrum infection on the right side following severe colds.

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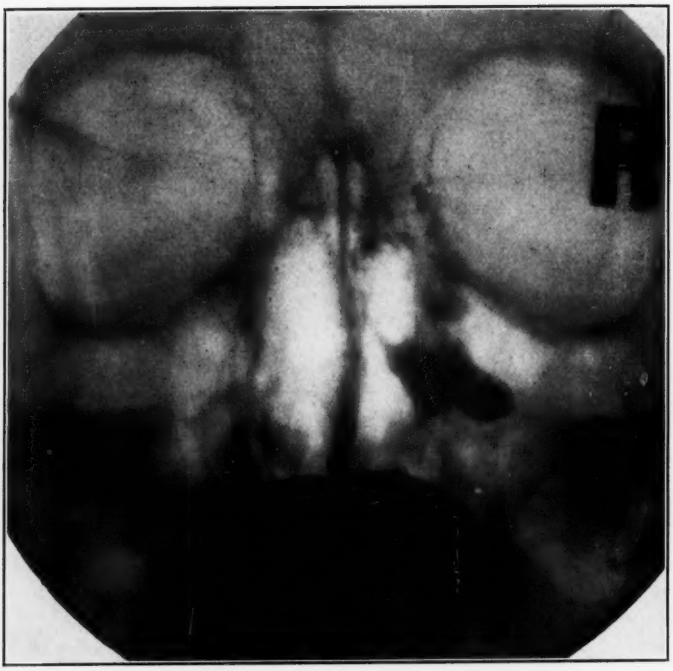


Fig. 1. Radiopaque in the right sphenoid, showing its conformation and a pneumatized pterygoid process.

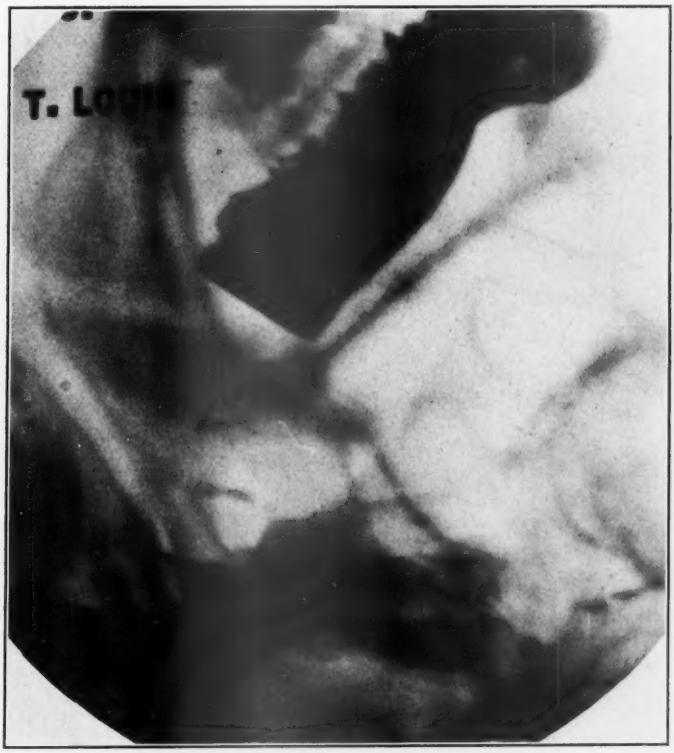


Fig. 2. Same filling as the previous figure. Patient in the inverted position (chin over ear). The radiopaque shows that the right cell occupies only the forward portion of the body of the sphenoid.

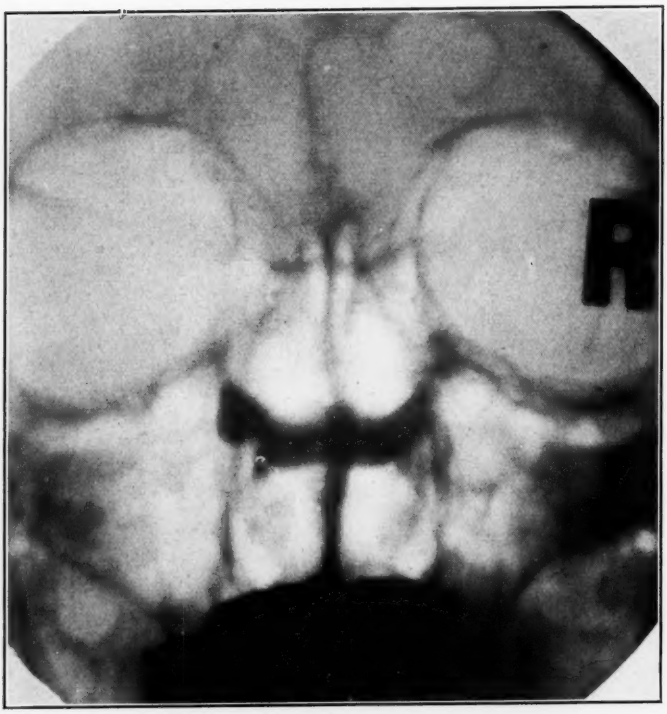


Fig. 3. Radiopaque in the left sphenoid. The oil was removed from the right sphenoid before this exposure was made. The radiopaque in this instance demonstrates the left sphenoid as traversing both halves of the body of the sphenoid bone.

Fig. 4. The same filling as in Fig. 3; the head in the inverted position. The left sphenoid sinus obviously occupies the posterior half of the body of the sphenoid bone.

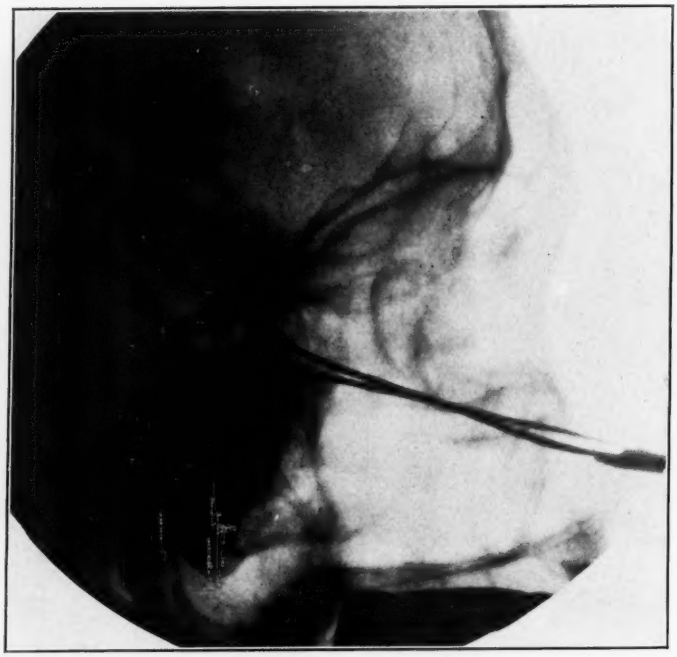


Fig. 5. Lateral view of the preceding. The curved cannula is in the left (filled) sphenoid. The straight probe is against the posterior wall of the right (unfilled) sphenoid.

LXXVI.

EXTRACTION OF BULLET FROM SPHENOID BONE.

SAMUEL SALINGER, M. D.,

CHICAGO.

A woman, age 46, was brought in to the Cook County Hospital with the history of having been shot through the jaw. There was some hemorrhage from the nose and mouth, arising evidently from the nasopharynx. A small wound of entrance was discovered on the right side of the face just behind the angle of the jaw; and there were numerous powder marks in the immediate vicinity. No wound of exit was found.

X-ray pictures disclosed a foreign body in the region of the sphenoid. It seemed to be to the left of the median line and just below the sphenoid sinus. An attempt was made to remove it through the nose but was unsuccessful. A short time later another attempt was made, more room being gained by removal of the septum, but on reaching the nasal aspect of the body of the sphenoid it was impossible, on account of bleeding and limited operative field, to locate the bullet.

A third attempt, in which the nose and lip were lifted through a sublabial incision, also resulted in failure. The patient left the hospital, still complaining of pain and headache, and six months later was brought to my office with the story of a fourth operation, which was likewise unsuccessful.

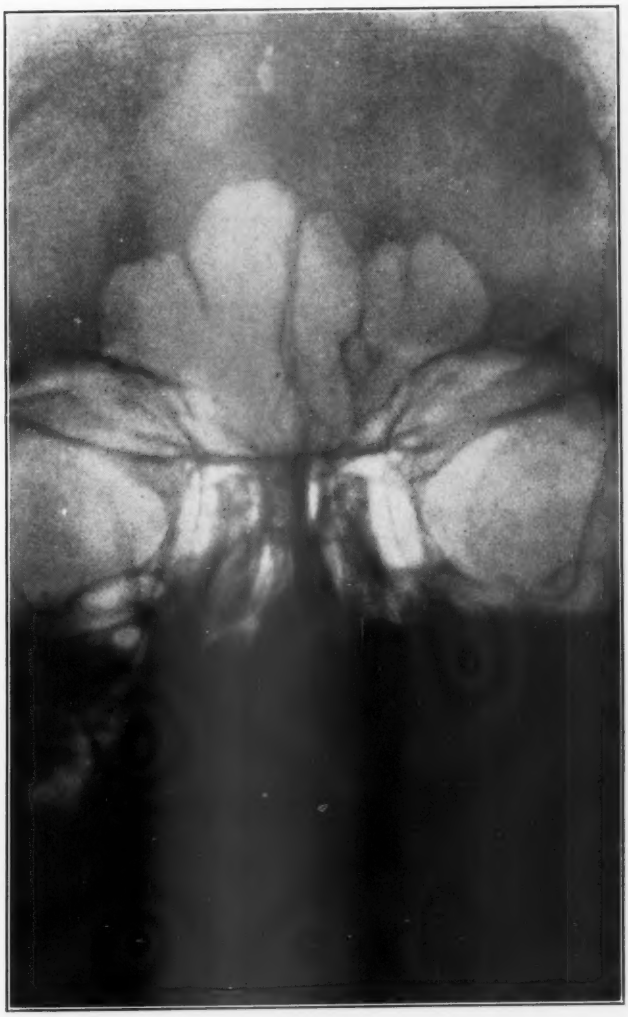
She was removed to the Michael Reese Hospital, where further roentgenologic studies, aided by a metal probe passed into the left side of the nose and held in place by packing, accurately localized the bullet within the body of the sphenoid on the left side at a point corresponding to the vault of the pharynx.

She was operated upon under general anesthesia July 29, 1930. The soft palate was split in the midline and retracted. The vault of the pharynx was incised and the soft tissues retracted, exposing the bone. A stiff probe found a small opening high up, which

was enlarged with a curette. The bullet was located and then exposed to view. It was slightly movable. Enough of the surrounding bone was removed with a curette and the bullet finally extracted by means of a hook and forceps. The wound in the pharynx was left open. The soft palate was repaired.

Recovery was uneventful.

25 East Washington Street.



X-ray picture disclosing a foreign body in the region of the sphenoid.

LXXVII.

STUDIES ON THE ACCESSORY NASAL SINUSES: THE COMPARATIVE MORPHOLOGY OF THE NASAL CAVITIES OF REPTILES AND BIRDS.

PAUL R. NEMOURS, M. D.,*

ST. LOUIS.

This study is the second in a series[1] of investigations in which an attempt is made to trace the phylogenetic history of the maxillary sinuses, the first being on the family of amphibia. For the convenience of the reader a brief classification of reptiles and birds adapted from Parker and Haswell[2] is included on the opposite page. Gegenbauer[3] gives a description of the nasal cavities of various reptiles and birds. He defines a true turbinate as a single free lamella-like projection of the skeletal wall into the nasal cavity in contradistinction to pseudoturbinates which are merely broad based projections into the nasal cavity, in which the skeletal wall may or may not take part. In the families of Lacertilia (lizards) and Ophidia (snakes), he describes a true turbinate. In the Chelonia (turtles) he is rather doubtful, but states that one of the lateral projections may be called a turbinate. In the Crocodilia (alligators) he describes one true turbinate as well as a pseudoturbinate. In the bird family he describes various numbers of turbinates, such as one for the pidgeon to three for the chicken and other species. However, in the case of the presence of more than one turbinate as in the chicken, only one, the middle turbinate, meets the definition of a true turbinate; the others being of the pseudoturbinate type.

Born[4] describes the nasal cavities and accessory structures of lizards, snakes, and birds, in several articles. He quotes Leydig extensively, who found Jacobson's organ, accessory nasal cavities, and the nasolacrimal duct in lizards and snakes. The structure of Jacobson's organ in the snake is described in detail by Mihal-

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kovics.[5] He believes the function of Jacobson's organ in a snake is to recognize the sex in other members of the same species. He points out the fact that Jacobson's organ communicates with the oral cavity by means of a narrow cleft, so that a rapid diffusion of gases would not be possible in order to test the contents of the oral cavity. He was struck by the fact that Jacobson's organ is very highly developed in crawling creatures. Where Jacobson's organ has a great development it has a sheltered location and its opening is narrow. This must be for a definite purpose. Seydel's designation of function for this organ as a sensory test of the expiratory air current as in amphibians and turtles does not seem likely. The exact substance that is perceived by this organ is not known. Mihalkovics accounts for this poor development in birds in that the eyes have taken over the function of determining sex in others of the same species. In the species of snake studied by Mihalkovics, there was described a pseudoturbinate, and a maxillary sinus. In describing the nasal cavities of turtles, he quotes Seydel who described the nasal cavities of both the land and water types. Both are similar, but the nasal cavity of the land type is more simple.

McCotter[6] studied Jacobson's organ in the turtle in the species Chrysemis punctata. He agrees with Seydel and Zuckerkandl in considering the anterior ventral epithelium which lies in a fossa as Jacobson's organ. He states that this epithelium is separated from the dorsal olfactory region by a thin streak of nonsensory epithelium. Zuckerkandl[7] described two divisions of the olfactory nerve, one to the dorsal region, and the larger to the two supplying the region of Jacobson's organ, or the region of the ventral olfactory epithelium. This was corroborated by McCotter. These two nerves proceed to different areas of the rhinencephalon. Mihalkovics considers the anterior end of the duct of the medial nasal gland as Jacobson's organ, stating that it is lined with a sensory type of epithelium similar to that found in mammals, also it received a good nerve supply. Zuckerkandl did not verify this, stating that the epithelium lining the duct was similar to that of a gland. Mihalkovics also describes the nasal cavities of the domestic chicken, the writer's specimen being similar in most respects to his description.

Rathke[8] gives a detailed account of dissections of young specimens of crocodiles and alligators. He found a maxillary sinus and several accessory nasal spaces derived from the posterior nasal space. The writer's specimen of a young alligator follows his description in most respects. Rathke apparently did not find Jacobson's organ in the family of crocodilia, but Rose[9] describes the formation of Jacobson's organ in the embryology of Crocodilus porosus. He describes, also, a pit in the vomer as the remains of this organ in the adult.

LACERTILIA (LIZARDS, ETC.)—(ANOLIS CAROLINENSIS).

The nasal cavities of the adult chameleon (anolis carolinensis) are surrounded by the following bones: the nasal, frontal, premaxilla, maxilla, and vomer; there is a cartilaginous septum with two thin lateral dorsal extensions above. The palate is primitive in type with the choanae of the nasal cavity leading directly into the oral cavity; within the palate there is located Jacobson's organ and the termination of the nasolacrimal duct. The external nares enter anteriorly and somewhat laterally into the nasal cavities which are round at this point, and in this region of the nasal cavity the epithelial lining is a nonciliated stratified type. The lateral nasal gland has its outlet here and is located laterally to the nasal cavities. Proceeding posteriorly, the nasal cavities extend laterally, the lateral nasal gland meanwhile assuming a more dorsal position. The epithelium has now changed to a different type. Laterally ciliated pseudocolumnar epithelium with goblet cells is present, which of course is typically respiratory epithelium, while on the medial side of the nasal cavities typical olfactory epithelium with Bowman's glands is seen. Toward the posterior end of the nasal cavities the choanæ are present, leading to the oral cavity from the lateral floor of the nasal cavities which end slightly posteriorly as blind cavities.

Jacobson's organ appears as a u-shaped structure beneath the ventral side of the anterior portion of each nasal cavity (Illustration 1). A thick layer of olfactory type of epithelium is present on the medial surface, and a thinner nonciliated stratified type on the lateral surface. Proceeding posteriorly this organ sends a lateral branch to the medial side of the maxilla where it meets and fuses with the nasolacrimal duct. The

posterior region and nasolacrimal duct are lined with a nonciliated stratified type of epithelium that is identical with the epithelium present on the lateral side of Jacobson's organ. Laterally there is another posterior extension or diverticulum of Jacobson's organ which ultimately unites laterally with the nasal cavity (Illustration 2). The epithelial lining of this part of Jacobson's organ is also a nonciliated stratified type.

The structure of Jacobson's organ in the chameleon varies from that described for other species of the lizard family. Communication with both oral and nasal cavities, and the emptying of the nasolacrimal duct between these openings is quite different from the descriptions of other lizards. Having two separate openings would allow a greater rapidity in diffusion of gases than with a single opening. Jacobson's organ may have importance in determining the contents of the oral cavity. The writer recalls only one other instance where the nasolacrimal duct enters Jacobson's organ, and that occurs in gymnophiona according to P. and F. Sarasin as quoted by Seydel.[10] No turbinates or nasal sinuses were found by the writer.

OPHIDIA (SNAKES).

Under the class of ophidia an adult oak snake (Elaphi obsoleta confinis) was studied. Anteriorly, the nasal cavities are partly surrounded by a thin cartilaginous skeleton, the cartilaginous septum sending lateral processes which surround the nasal cavities except on the ventrolateral portion. The bones surrounding the nasal cavities are the nasal, premaxilla, maxilla, frontal, palate and vomer. The nasal cavities extend slightly anteriorly to the external nares which enter laterally. At this point the nasal cavities have a circular form. Proceeding posteriorly the nasal cavities become somewhat oval in shape and two projections are seen encroaching on the nasal space; one at the junction of the septum and nasal floor, and the other from the medial lateral nasal wall. The former serves to enclose Jacobson's organ, while the latter is a turbinate. This structure extends into the nasal cavity, dividing it into an upper olfactory and lower respiratory region; and seems to be an extension of the lower lateral cartilaginous nasal wall. Further back the

turbinate and Jacobson's organ disappear, with the nasal cavities becoming smaller, and finally they unite and empty in a single choana.

The epithelium at the external nares is squamous in type, further in the olfactory type appears, but as one proceeds posteriorly, respiratory type (ciliated pseudostratified columnar) appears on the floor and partially on the lower lateral nasal wall. The turbinate and the remainder of the wall of the nasal cavity has olfactory type of epithelium, while posterior to the turbinate the epithelium is entirely respiratory in type.

The turbinate is a true one, meeting the definition given by Gegenbauer[3] in that it is a simple free lamella-like projection into the nasal cavity from the skeletal wall. A well developed lateral nasal gland is present, having its outlet entering the posterior wall of the external nares. It lies lateral to the nasal cavity, and in its more posterior portion is incorporated in the body of the turbinate (Illustration 3).

No evidence was found of an accessory nasal sinus in the writer's specimen. The palate of the snake is primitive in type, and a single choana is present leading into the oral cavity. In some specimens of snakes each nasal cavity terminates in a secondary choana lying laterally, this in turn leads to the single choana in the center of the palate.

Jacobson's organ reaches one of its greatest developments in the snake family and is located just lateral to the septum at its lower point. It is located above the premaxilla, medially to the intermaxilla, and above the vomer. The paraseptal cartilage projects into the lumen of Jacobson's organ, giving the appearance of a knob-like projection (fungiform eminence). The excretory duct is in the form of a narrow cleft opening into the oral cavity close to the choana. The cavity of Jacobson's organ has the appearance of a quarter moon. The epithelial lining on the fungiform eminence is thin nonsensory respiratory in type; however, on the other side it is enormously thickened.

There is a superficial layer on the external surface which has the same structure as ordinary olfactory epithelium — that is, olfactory and supporting cells. The deeper layer is much thicker and seems to be arranged somewhat in the form of cords by

means of nerve fibers, giving the appearance to some extent of parathyroid tissue. By special staining Mihalkovics found a rich capillary network between these two layers. No excretory ducts were seen; the cells probably do not secrete, but are modified nerve cells similar to small granule cells of the cerebellum. The shape of these cells is round with little cytoplasm and give the writer the impression of having an olfactory function, for Jacobson's organ is richly supplied with nerves from Jacobson's nerve, a branch of the olfactory. Toward the posterior end of the turbinate a small recess is seen in the septum ending blindly on the anterior side, which is lined with respiratory epithelium. The nasolacrimal duct empties into the oral cavity close to the opening of Jacobson's organ and it also is lined with respiratory epithelium.

CHELONIA.

Under the class of Chelonia, an adult turtle (Pseudemys elegans) was studied. The external nares enter the nasal cavities directly anteriorly, the cartilaginous skeleton first being above and below; further posteriorly the nasal cavities have a complete cartilaginous investment, perforated only for such structures as the olfactory nerve and ducts of glands. The name given to it is the annular nasal cartilage; this in turn is surrounded by a complete bony investment. The turtle has a true hard palate, since the palatal processes of the maxilla come in apposition. There is no fusion of the bones, however. A thin layer of connective tissue or periosteum lies in between them, which is also true for other bones of the turtle skull.

Beginning at the external nares, the nasal cavities are round and are lined with a stratified type of epithelium; the form of the cavities soon changes to oval, and a diverticulum appears ventrally lined with olfactory type of epithelium. (Illustration 4.) The epithelium of the nasal cavities has meanwhile changed to an olfactory type, Bowman's glands not being present, however. A small diverticulum can also be seen entering the roof of the nasal cavity, this being also lined with olfactory epithelium. The duct of a well developed gland located medially and above the nasal cavities empties into this diverticulum.

In the anterior part of the septum, a round duct can be seen which enters the nasal cavity, the epithelium appearing to be a thin olfactory type which extends along the entire length of the duct as it proceeds posteriorly until it changes to a glandular type where several ducts of the medial nasal gland appear. This is a small gland in comparison with the one located above the nasal cavity. Seydel considered this to be the gland of Jacobson's organ located on the floor of the nasal cavity. The writer could note corroborate Zuckerkandl's observation that the epithelium of the duct of the medial nasal gland as being similar in type to that of the gland itself.

As one follows the nasal cavities posteriorly, they have a marked lengthening of the dorsal ventral axis, the epithelium now has changed to the usual type of olfactory epithelium with Bowman's glands. One sees a medial and two lateral projections from the nasal walls extending into the nasal cavity, which is thereby somewhat subdivided into three divisions. The turtle does not have a true turbinate, however; there is no projection of a lamellar structure extending from the skeletal wall into these projections. Respiratory type of epithelium first makes its appearance in a small area just above the medial nasal projection. Slightly posterior to this one can see a small area of respiratory epithelium opposite on the lateral nasal wall. Soon the upper part of the nasal cavity ends as a blind pouch; the lower part of the nasal cavity extends laterally. At this point another diverticulum is seen entering the nasal cavity medially and above. It is lined with respiratory epithelium, and is considered by Mihalkovics to be a maxillary sinus. The nasal cavities assume a more circular form and extend posteriorly to the choanae as the nasal ducts. Respiratory type of epithelium is located here exclusively; this is somewhat different from the usual respiratory epithelium in that several layers of cells are located basally; above them there is a thick layer of mucous cells with occasional nuclei interspersed between. Cilia are not so well outlined. No evidence was found of a nasolacrimal duct.

CROCODILIA.

Under the class of Crocodilia, a young specimen of an alligator (Alligator Mississippiensis) 15.5 cm. in length was studied.

The skull of the alligator is complicated. Those interested are referred to texts on comparative anatomy. It is of interest that the alligator has a true hard palate of the definitive type. The palatal processes of the maxilla and premaxilla come in opposition and fuse.

The nasal cavities can be divided into anterior and posterior portions with the division at about half the length of the nasal passageway. They are divided by a cartilaginous septum which sends upper and lower lateral processes which surround the nasal cavities almost entirely. The anterior end of the nasal cavities begin at the external nares which enter the nasal cavities dorsally at the tip of the snout and proceed posteriorly in a broad but not very high space. The floor gradually sinks as one proceeds posteriorly, and the second half of the nasal cavity appears with a bony lamella extending from the septum to the lateral wall which divides the posterior portion into an upper and lower portion. The lamella is composed of palatine and sphenoid bones, and has no opening for communication. The upper space is thereby formed into a blind pouch-like cavity, while the lower part conducts the inspired air to the choanae. This lower part is a simple structure and is designated as the nasal duct. The upper cavity is somewhat complicated, however.

At the point where the nasal passage goes into the nasal duct a turbinate appears above at the beginning of the upper part of the posterior nasal space. It arises from the roof of the nasal cavity and extends posteriorly about two-thirds of the length of the blind space. It meets Gegenbaur's[5] definition of a turbinate since it is a simple lamella-like structure arising from the skeletal wall of the nasal cavity. Behind the turbinate there is a convex bulge of the lateral nasal wall into the upper nasal cavity, partly covered anteriorly by the turbinate. (Illustration 6.) It is a hollow structure and was designated by Rathke[6] as the posterior outer olfactory turbinate; Gegenbauer classifies it as a pseudo-turbinate. It communicates anteriorly with the nasal cavity by means of a small duct beginning at its posterior end and extending forwardly along the floor to a space beneath the true turbinate. Just anterior to the true turbinate there is another structure which is hollow, and projects somewhat laterally, but does not encroach

on the nasal space. It was designated by Rathke as the anterior outer olfactory turbinate. However, it is not a true turbinate and is not visible from a medial view, but empties into the nasal cavity. All of these structures have cartilaginous walls, bone not being present. The writer will refer to the two pseudoturbinates as the anterior and posterior nasal pouches. Between them and arising just about the region where the nasal duct begins there is an outpouching of the nasal mucosa laterally outside of the cartilaginous skeleton which proceeds anteriorly between the two nasal pouches.

The nasolacrimal duct passes dorsally and laterally to the nasal cavity as it proceeds anteriorly, and empties into the nasal cavity behind the opening of the maxillary sinus. The maxillary sinus in a young alligator (Illustration 5) opens directly into the nasal cavity, but there seems to be a turning of the sinus beneath the floor of the nasal cavity in the adult and the sinus communicates with the nasal cavity by means of a canal which proceeds posteriorly and ends at about the beginning of the nasal duct.

The alligator has a large lateral nasal gland which begins about the center of the nasal cavity and goes forward medially to the nasolacrimal duct and terminates on the posterior wall of the external nares. The gland is outside of the cartilaginous nasal capsule.

The writer did not find any trace of Jacobson's organ in a young or adult form of the alligator. However, Rose[7] gives evidence of a rudimentary Jacobson's organ in the embryo of Crocodilus Porusus at the beginning of the nasal duct and shows the picture of a skull of an adult Crocodilus Porosus with a pit in the vomer.

The epithelium at the external nares is squamous, but once inside the nasal chamber it changes to the olfactory type of epithelium with Bowman's glands. Then respiratory type of epithelium (ciliated columnar) appears on the lateral wall and floor of the nasal cavity, while the maxillary sinus is also lined with epithelium of this type. The nasolacrimal duct is a very glandular structure, and is lined with respiratory type of epithelium. The same type of epithelium lines the nasal duct and the pouch of the nasal mucosa between the nasal pouches. The entire mucous membrane

of the upper nasal space above the nasal duct is olfactory except the floor. The true turbinate has olfactory epithelium which is also the case for the anterior nasal pouch. The mucosa inside the posterior nasal pouch is a thin respiratory type.

AVES.

As a representative of the birds a series of frontal section of a newly hatched chicken (Gallus domesticus) was studied. Birds as a group have varied numbers of so-called turbinates, varying from one to three. The external nares communicates with the nasal cavity through a narrow cleft which is covered to some extent medially by a special structure of the anterior part of the nose, the vestibular concha which deflects the air current. This is a peculiar structure having its base near the roof of the nasal cavity, and anteriorly has the appearance of a true turbinate. However, at its posterior end it extends over medially and joins the septum, forming the roof of a small accessory nasal cavity in the anterior part of the palate ,the inferior nasal recess. Further posteriorly one sees the middle turbinate which meets Gegenbauer's definition of a true turbinate. It is a lamella-like structure having a roll of one and one-half times (Illustration 7). This turbinate corresponds to the inferior turbinate of man, being entirely covered with respiratory type of epithelium, and having several structures in somewhat the same relationship to it.

Birds are very closely related to the reptiles in their development and anatomic characteristics, such as the arrangement of the skull, palate, etc. The middle turbinate of the chicken, however, does not exactly correspond to the turbinates found in lacertelia, ophidia, and crocodilia, because the turbinates in the reptiles are covered partially or entirely with olfactory epithelium and apparently serve to increase the olfactory area. The superior turbinate of birds corresponds more closely to the turbinates of lizards, snakes, and alligators.

In the chicken the nasolocrimal duct enters the nasal cavity beneath it, the accessory sinuses above (Illustration 8). As one approaches the posterior end of the middle turbinate, the superior turbinate is seen to appear as a round bulging structure from the

upper lateral nasal wall and is at the same level as the orbit. This turbinate corresponds somewhat to the turbinate of snakes and lizards and crocodiles. The palate of the chicken is of the primitive type forming a roof over the nasal cavity to about the center of the middle turbinate and contains the palatine glands and the inferior nasal recess. The maxilla is seen here; anteriorly to the maxilla, the premaxilla is located.

Posteriorily the palate is divided into a Y-shaped affair, the middle palatine fissure corresponding to that of the reptiles, while the two shelf-like processes are formed by extensions of a secondary palate.

The chicken has well developed accessory nasal cavities. There is a large cavity on the medial side of the maxilla which extends forward medially to the premaxilla as the palatine sinus. Posteriorly, the maxillary sinus extends beneath the orbit as the orbital sinus. The maxillary sinus enters the nose above the middle turbinate.

There is a well developed lateral nasal gland which lies laterally to the nasal cavity at its upper part and extends forward and crosses medially in the palate just behind the inferior nasal recess; then proceeds forward a short space and then travels upward in the septum and terminates in the nasal cavity. It corresponds to the lateral nasal gland of reptiles.

The nasal skeleton is cartilaginous supported with the nasal maxilla, premaxilla, frontal and other bones. The skull of birds is a rather complicated structure.

The nasolacrimal duct is rather large, having its nasal end beneath the middle turbinate and is lined with respiratory type of epithelium.

The epithelium of the greater part of the nasal cavities is pseudostratified ciliated columnar epithelium. It has numerous pockets extending to the base of the epithelium consisting of mucous cells. The sinuses are lined with a thin columnar type of epithelium. Olfactory epithelium is limited in extent, being situated posteriorly in the nose on the superior turbinate and upper septum and nasal roof. Bowman's glands are poorly developed.

No true Jacobson's organ was found. Mihalkovics[5] studied several species of birds without apparently finding it. He was struck by the fact that this organ is highly developed in some reptiles and apparently missing in birds which are closely related. However, he points out the fact of the close resemblance of the epithelium of the terminal part of the duct of the lateral nasal gland to the olfactory epithelium and its similarity to Jacobson's organ in the higher vertebrates. He believes this is a form of Jacobson's organ.

SUMMARY.

The occurrence of the maxillary sinus varies in reptiles. Some members of the Lacertelia (lizards) have one, others have not; in none, however, is the sinus developed to any great size. In the Ophidia (snakes) again, there is a variation present in that some species have them and others do not. Apparently in this group also, there is no great development of the sinus. The Chelonia (turtles) have a small maxillary sinus. The crocodile family (alligators) has the greatest development of the maxillary sinus, attaining a length of 7.5 to 10 cm. and a width of about 7.5 cm. and a height of 1 to 1.5 cm. in the adult. In addition to the maxillary sinus, the alligator has several other accessory nasal cavities derived from the upper posterior nasal space. It is of interest that the anterior nasal pouch or anterior outer olfactory turbinate of Rathke is lined with olfactory epithelium.

In birds there is a well developed accessory nasal space or maxillary sinus on the medial side of the maxilla and premaxilla. It is of interest that this sinus empties above the middle turbinate of the chicken, which corresponds to the maxilloturbinal of mammals or the inferior turbinate of man. In a study of the vertebrate forms beneath the mammals, the closest approach to a maxillary sinus such as we have in man, for example, is that of the alligator. It is completely surrounded with bony walls of the maxilla and connects with the nasal cavity by means of a narrow duct. All of the maxillary sinuses of the lower forms are lined with respiratory type of epithelium.

The incidence of true turbinates meeting Gegenbauer's definition varies in reptiles and birds. Some of the lizards and snakes

have one, others do not. Apparently the turtle family has none, the alligators and birds have one. In no form was more than one turbinate found; the others, if present, were pseudoturbinate in type. In none of the forms other than those of mammalia have bony lamella of the ethmoid been found; the others have cartilaginous projections from a cartilaginous skeletal wall. Nothing was found by the writer suggestive of cavernous or erectile tissue in reptiles or birds. In the turtle and alligator, which have the hard or definitive type of palate, there wasn't any suggestion of a nasopharynx, the choanae leading directly into the oral cavity.

The nasolacrimal duct is absent in turtles; it is present, however, in the other reptiles, emptying into the oral cavity in close relationship with Jacobson's organ, or directy into it in the lizards and snakes. In alligators and birds it empties into the nasal cavity. It is of interest that the nasolacrimal duct of the chicken empties into the nasal cavity beneath the middle turbinate, which corresponds to the inferior turbinate of man.

A lateral nasal gland is present in all of the reptiles, emptying into the external nares or close to it (chameleon and turtle). In birds this duct passes medially beneath the floor of the nasal cavity and terminates in the medial wall or septum of the nasal cavity. The lateral nasal gland is also present in the amphibia and various species of mammals. It has not been found in the human, however. Bast[11] takes up the structure of the lateral nasal gland of dogs and finds it has a location close to the maxillary sinus, but its duct empties into the vestibule of the nose more than 5 cm. from the maxillary sinus. He states that it may, together with the alveolar serous glands of the medial wall and certain modified nerve cells in the epithelium of the maxillary sinus, offer functional evidence. These structures are not found in the less developed maxillary sinus of man.

The development of Jacobson's organ varies. In alligators it is very rudimentary or absent; in birds and turtles it is poorly developed, but reaches one of its greatest developments in lizards and especially in snakes. Here the organ is of huge size. The epithelium is arranged somewhat in a cordlike structure, suggestive of parathyroid tissue. The cells are apparently a modified nerve cell type. Since no ducts were found, it probably has no

glandular function such as Bowman's glands in olfactory epithelium. The writer does not know of any work demonstrating internal secretory activity for these cells. Mihalkovics[3] attributes the great development of this organ to the necessity for recognition of sex in other members of the same species. He was led to this conclusion on account of the great development of this organ in crawling forms of vertebrates. As a purely gustatory organ its opening is probably too small for a rapid diffusion of gases. He also attributes the lack of its great development in birds as an indication that the eyes have taken over the function of recognition of sex in other members of the same species. In support of this he mentions that in fishes and birds there is apparently a rudimentary olfactory development, and that the eyes take care of the recognition of sex. He does not believe Jacobson's organ has the same function as olfactory epithelium, for a differentiation from the usual olfactory epithelium would be superfluous, and unnecessary organs become rudimentary or disappear.

Classification of Reptiles and Birds.

REPTILES.

Order 1. Squamata.—Sub-order a: Lacertelia. As a rule limbs are present and are adapted for walking. Movable tympanum. Includes all lizards, monitors, iguanas, and chameleons.

Sub-order b: Ophidia. Long narrow body devoid of limbs. No movable tympanum. Includes all of the snakes.

Order 2. Rhyncocephalia.—Well adapted limbs for walking. Lizard-like reptiles. Only one living species, the Hatteria or Sphenodon of New Zealand.

Order 3. Chelonia.—Reptiles having the body enclosed in bony plates. Includes all of the tortoises and turtles.

Order 4. Crocodilia.—Reptiles in which the dorsal, or both dorsal and ventral surfaces, are covered with rows of sculptured scutes supporting horny scales.

Aves or Birds.—Includes all of the numerous families of birds. Those interested in a detailed classification are referred to Parker and Haswell.

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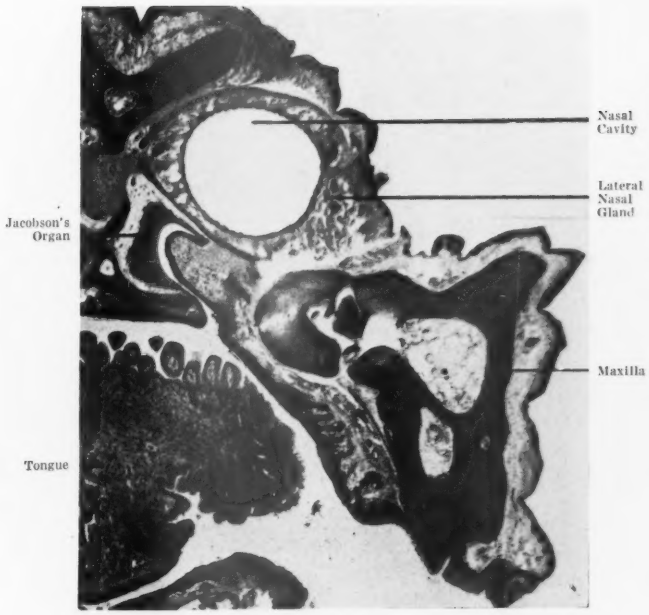

Illustration 1.
Chameleon.

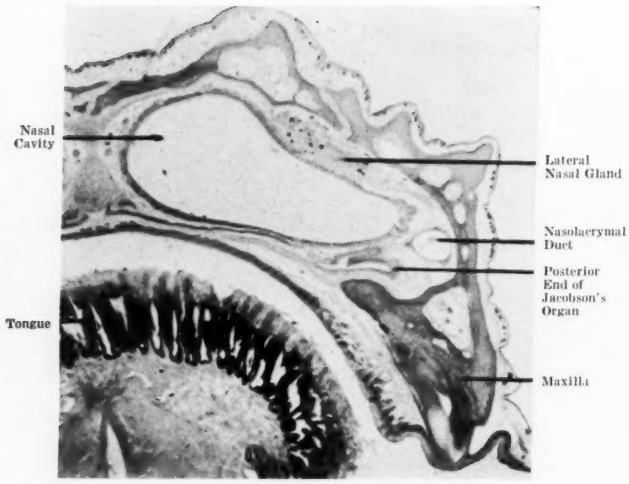

Illustration 2.
Chameleon.

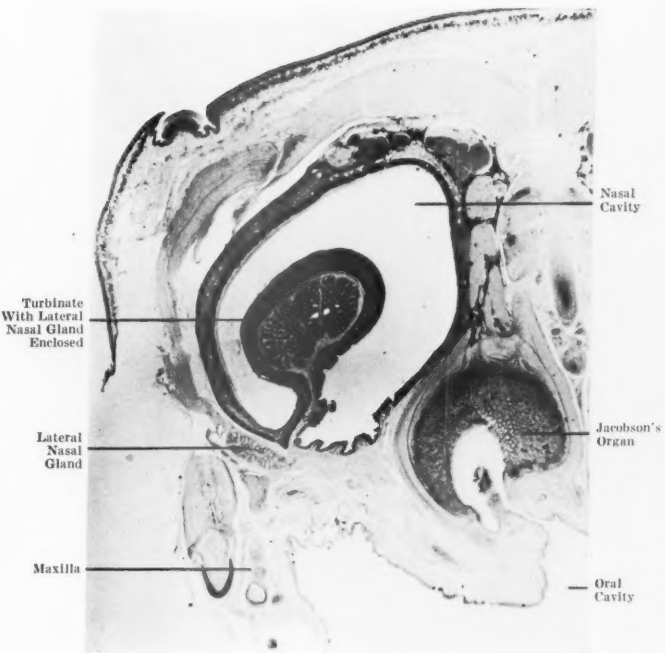

Illustration 3.
Oak Snake.

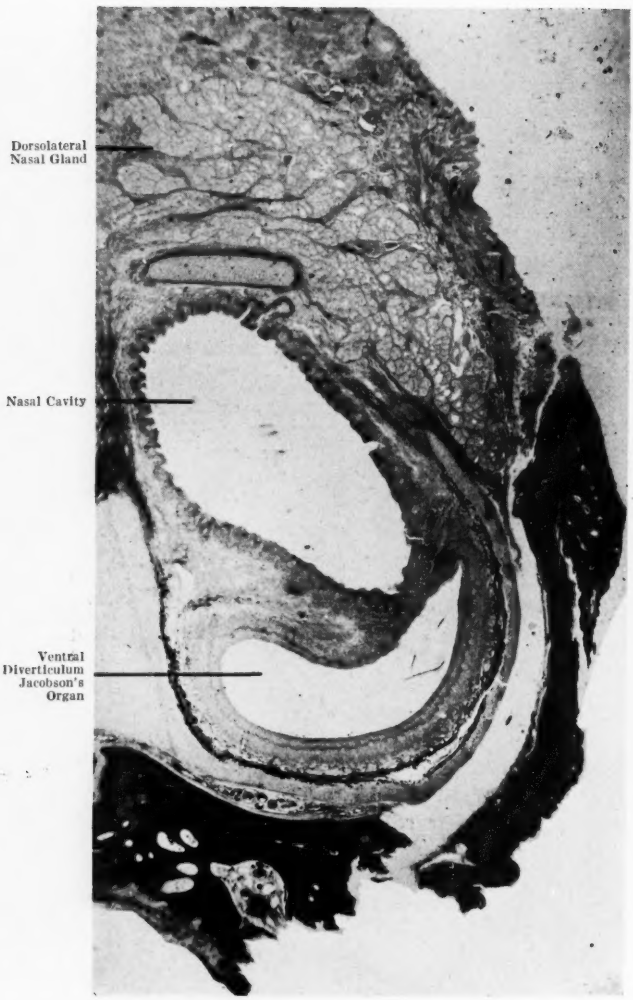

Illustration 4.
Turtle.

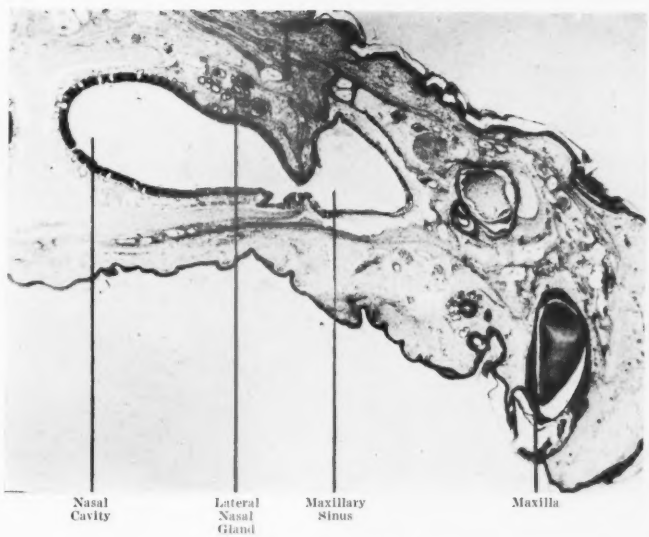

Illustration 5.
Alligator.

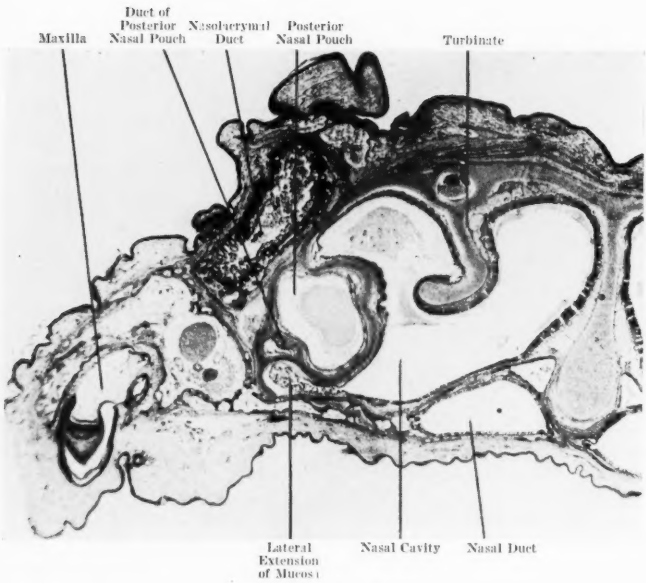

Illustration 6.
Alligator.

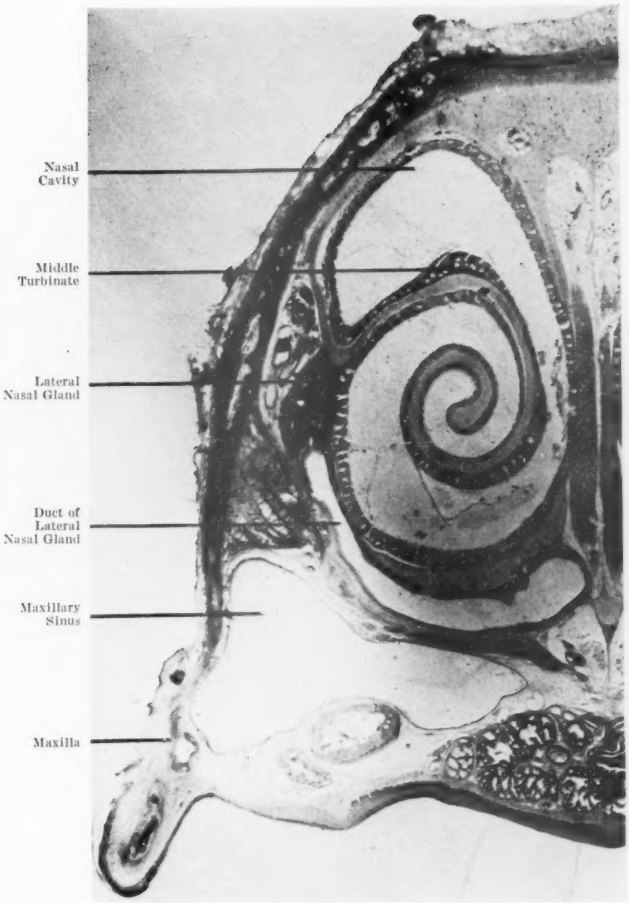

Illustration 7.
Chicken.

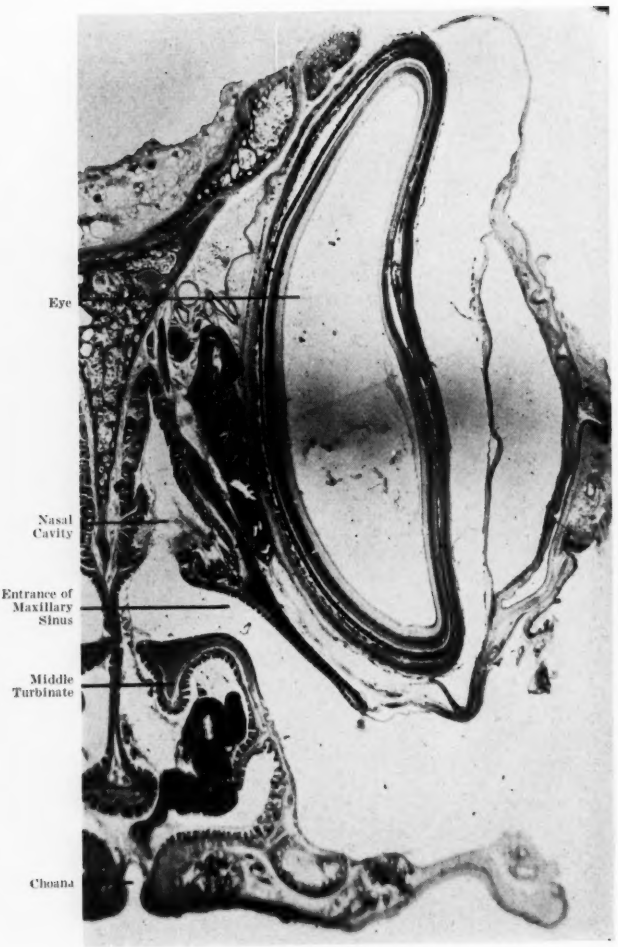

Illustration 8.
Chicken.

LXXVIII.

A NEW LIGHT ATTACHMENT FOR SYRINGE TO BE USED FOR THE INJECTION OF IODIZED OIL AND MEDICAMENTS INTO THE LUNG BY THE ASPIRATION METHOD.

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ST. LOUIS.

A new light attachment for a syringe is presented. This attachment consists of an ordinary inexpensive light bulb (3), is covered by a removable hood (2) and can be made to connect (4) to any type of 20 cc. syringe. In the illustration it is seen attached to a 20 cc. asepto Luer-"Lok" syringe. The battery (7) is of the ordinary flashlight type, with cord (6). It can be attached to a pocket by a hook (8) or hung on the wall. The entire light connection is simple and is so made as to rotate on the syringe. Light can thus be directed wherever desired. All the parts can be sterilized in the usual manner. The bulb, an ordinary nonsurgical one, rests on the syringe, does not get hot, is inexpensive, lasts for quite a time and does not require the delicate care which must be given a surgical lamp.

Only a 20 cc. syringe (5) with a straight cannula (1) is necessary to introduce iodized oil into the tracheobronchial tree according to the aspiration method recommended by Singer (J. A. M. A., October 16, 1926, 87:1298).

Illumination during the performance of the injection of a substance into the bronchial tree has usually been obtained from some sort of hand light held by an assistant. This kind of illumination leaves something to be desired. The light described above has been found to have decided advantages in this connection. It can also be used for the injections of various substances into the lung and elsewhere.

*Chest service of Barnes Hospital and Department of Surgery of Washington University School of Medicine, St. Louis.

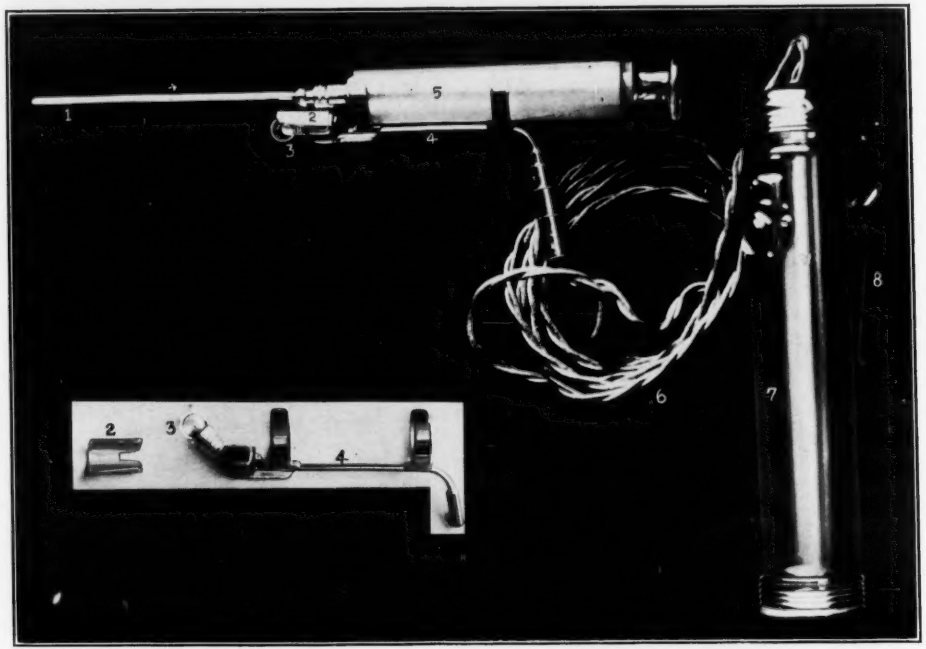

Fig. 1.

LXXIX.

LUNG IMMOBILIZATION IN THE TREATMENT OF PULMONARY TUBERCULOSIS AND ITS INFLUENCE ON THE LARYNX (A FURTHER REPORT.)*

JULIUS P. DWORETZKY, M. D.

LIBERTY, N. Y.

In discussing lung immobilization in the treatment of pulmonary tuberculosis and its effects on the larynx, reference will be made only to three principal procedures, the basic principle of which consists of the application of mechanical and physiologic rest through the compression of the diseased lung.

The first procedure, and the one most commonly employed, is artificial pneumothorax. This consists of inflating the pleural space of the affected or the more affected lung with air, compressing it for the purpose of securing necessary rest. This form of treatment may be beneficially employed in about 10 to 15 per cent of all cases of pulmonary tuberculosis, and is especially indicated in those cases where the disease is active and progressive, where the active lesion is confined to one lung, and where the hygienic-dietetic treatment has been given a fair trial and failed to bring about the desired result.

The second procedure, which is now quite frequently employed, is phrenicotomy, or phrenic evulsion, which consists of either severing or evulsing the phrenic nerve on the affected or more affected side, through a small incision above the clavicle. The purpose of this procedure is twofold. First, to relax the diaphragm, thus aiding mechanical fixation of the lung. Second, to cause partial compression of the lung through the elevation of the diaphragm, which usually occurs as a result of this operation. This procedure is indicated in cases where, for one reason or another, artificial pneumothorax cannot be induced, and may be employed either as the sole treatment or as adjunctive treat-

*Read before the American Laryngological, Rhinological and Otological Society at its annual meeting in Atlantic City, N. J., May 28, 1930.

ment to artificial pneumothorax or preceding a thoracoplastic operation.

The third commonly used measure is extrapleural thoracoplasty. This surgical procedure is usually employed in the same type of case where artificial pneumothorax is indicated, but where the latter cannot be induced on account of extensive adhesions. In this operation segments of the ribs of one side of the chest are removed extrapleurally, and the purpose is mainly to obliterate cavities by compressing the diseased lung and thereby applying rest to the affected areas.

In 1925 I had the privilege of presenting a paper before the Triological Society on "Artificial Pneumothorax in the Treatment of Pulmonary Tuberculosis and Its Effects on the Larynx."[1] In that paper I reviewed the subject of artificial pneumothorax as applied in pulmonary tuberculosis, briefly outlining the technic and the modus operandi. I then stated that in going over the records of my patients treated by artificial pneumothorax I was impressed with the fact that in not a single instance where artificial pneumothorax was successfully employed did laryngeal tuberculosis occur as a complication. It was also my observation at that time that several of my patients who received artificial pneumothorax for their pulmonary lesion, and who also presented a coexisting laryngeal lesion, showed a definite improvement in the larynx. This was especially true of the subacute cases of laryngeal tuberculosis. Under this adjunctive treatment, I observed that infiltrations of the larynx were absorbed, granulations disappeared and ulcerations healed. This was, of course, in accordance with the general understanding that when the pulmonary lesion improves, the laryngeal lesion improves also.

I then presented two tables, one of which showed 1,592 cases of pulmonary tuberculosis, treated by various men with artificial pneumothorax, and only four of these cases developed tuberculous laryngitis. Of the four, two were reported by Dr. Lawrason Brown, of Saranac Lake, and he stated that it was quite probable that the laryngeal lesion existed prior to the time artificial pneumothorax was administered.

The other table showed thirty-two cases of laryngopulmonary tuberculosis, treated by artificial pneumothorax, of which twenty-

six were definitely improved, four remained stationary and two died. The four stationary cases, according to the report of Loomis Sanatorium, did not have a satisfactory collapse of actively secreting cavities.

The summary of the paper then was that:

1. Artificial pneumothorax successfully employed in properly selected cases will prevent the development of tuberculosis of the larynx in 99.5 per cent of the cases of pulmonary tuberculosis thus treated.

2. Artificial pneumothorax is very valuable in the treatment of suitable cases of laryngopulmonary tuberculosis, and when applied promptly and efficiently improvement must follow in the larynx as well as in the lungs.

Five years have now elapsed since the preliminary report of my first observation on the effects of pneumothorax on the larynx, and it seemed to me that it might be of interest to this society if I gathered the more recent views on this subject for the purpose of comparison. I have, therefore, again communicated with a number of observers throughout the country and also with several men abroad, obtaining their opinions, and, where possible, some statistical information on the subject.

Before discussing the effects of lung immobilization on tuberculous laryngitis, I wish to state that tuberculosis of the larynx must always be viewed as a complication of pulmonary tuberculosis and never as a disease entity. It is now generally admitted that tuberculous laryngitis is a surface infection, and only in very rare instances does the larynx become infected through the lymph or blood circulation.

It is my personal opinion that tuberculous laryngitis is never a primary infection, and I feel that in cases where laryngeal tuberculosis has been reported as primary, if we search diligently the original source of infection will be found in the lungs or elsewhere in the body.

It has also been my observation that laryngeal tuberculosis occurs only in active cases of pulmonary tuberculosis and nearly always in those cases which have a positive sputum, but never develops in cases which have become definitely arrested.

In summarizing the pathogenesis of tuberculous laryngitis, it may be stated, therefore, that tuberculosis of the larynx is a surface infection, always secondary to pulmonary tuberculosis or to tuberculosis elsewhere in the body, and occurs only in active cases and usually in those that have a positive sputum, but never in cases after an arrest has been definitely attained.

In order, therefore, to prevent laryngeal tuberculosis, it behooves us to bring about an arrest of the active pulmonary lesion. Artificial pneumothorax, in properly selected cases, is an admirable means to bring about such a desired result, and when the collapse is satisfactory it will not only prevent the development of tuberculous laryngitis but it will also exert a beneficial influence on an already existing laryngeal lesion.

The following table will summarize the gathered statistics showing the part artificial pneumothorax plays in the prevention of tuberculous laryngitis:

TABLE NO. 1.

Name	Number of Pneumothoraces	Number Developing Laryngitis	
I. D. Bronfin, Denver	450	2	Manifestations of miliary Tbc.
Herman H. Cole, Springfield, Ill.	500	6	Secondary to Tbc. in abdomen.
Geo. T. Evans, Hopemont, Va.	80	0	
Ralph C. Mason, Portland, Ore.	1500	1	Rapidly progressive fibrocaseous - cavernous type with dissemination in contralateral lung.
LeRoy Peters, Albuquerque	800	0	
W. L. Rathbun, Cassedaga, N. Y.	72	0	
Grant Thorburn, New York	150	0	
B. P. Stivelman, New York	108	1	Discontinued treatment against advice.
Henry Boswell, Sanatorium, Mo.	15	0	
Victor F. Cullen, Baltimore	105	0	
M. Pollak, Peoria, Ill.	100	0	
P. H. Ringer, Asheville	500	0	
Foster Murray, Brooklyn	250	0	
Mary L. Hamblett, Rhode Island	77	1	May have occurred before treatment.
Chas. Giese, Colorado Springs	175	1	(?) Called unsatisfactory collapse.
Chas. Atkinson, Banning, Cal.	200	0	
R. A. Bendove, New York	300	0	
	5382	12	

From the above table it will be observed that out of 5,382 cases of pulmonary tuberculosis treated by artificial pneumothorax, only twelve cases developed tuberculosis of the larynx. Of these, the two cases reported by Dr. Bronfin occurred in patients who developed miliary tuberculosis. In the case of Dr. Giese, the collapse was unsatisfactory. In the single case of Dr. Matson, there was a rapidly progressive fibrocaseous-cavernous type of pulmonary tuberculosis with dissemination in the contralateral lung. In the case of Dr. Hamblett, according to the doctor's statement, the lesion may have been there before the artificial pneumothorax was instituted, and in the case of Dr. Stivelman it occurred when the artificial pneumothorax was discontinued against advice. In the six cases reported by Dr. Cole, it was the doctor's opinion that they were probably secondary to tuberculosis of the abdomen.

This, to my mind, is very striking, especially when we consider the fact that artificial pneumothorax is usually administered in cases of the active and progressive type of pulmonary tuberculosis, mainly in the advanced or rapidly advancing cases, where a great deal of destruction of lung tissue has taken place, with softening and cavitation; where the symptoms, such as cough, expectoration or hemoptysis are troublesome, and where simpler methods have been given a fair trial and have failed. Cases of this particular type, in my experience, if allowed to run an unabated course, often develop tuberculosis of the larynx.

In this connection, I can plainly recall the case of a man, age 29, who first consulted me on October 15, 1929. He gave a history of tuberculosis dating back three years, complained of troublesome cough, expectoration, fever, malaise and recent hemoptysis. Physical examination, confirmed by fluoroscopy and X-ray, showed an extensive involvement of the entire left lung with cavitation at the left upper lobe. The right lung was fairly clear. The larynx was negative. Artificial pneumothorax, to be followed possibly by phrenicotomy, was advised for the purpose of compressing the left lung. Patient refused to accept this form of treatment and was not heard from until January 20, 1930, when he again came to my office, this time complaining of severe

dysphagia. Laryngoscopic examination revealed an acute infiltration of the right side of the epiglottis, also of the right arytenoid. The pulmonary lesion was considerably more active. Dysphagia was so severe that I had to resort to blocking the right superior laryngeal nerve with alcohol. This gave him relief for about three weeks, when the entire epiglottis became involved, with the formation of ulcers. I then blocked the left superior laryngeal nerve, again with temporary relief. The fever, however, continued very high. Emaciation progressed quite rapidly, with loss of about 26 pounds in weight in four months, and the patient died April 1, 1930, at St. Anthony's Hospital, Woodhaven, L. I. Had this patient accepted the offered treatment, I feel that the activity of the lesion could have been checked, the larynx would not have become infected, and the patient's life most likely would have been spared.

The fact that the occurrence of tuberculous laryngitis is so rare among satisfactory pneumothorax cases can only be attributed to the salutary effect which artificial pneumothorax exerts on the pulmonary lesion. By reason of the compression and immobilization of the affected lung, first, we eliminate or at least reduce the original source of infection, thus avoiding the constant passing of bacilli laden sputum over the laryngeal mucosa. Second, by affording relief from the cough, the lessening or disappearance of which usually follows a successful pneumothorax, we avoid a great deal of local trauma to the larynx and thus prevent the formation of a lowered local resistance. Third, by reducing the debilitating symptoms of toxemia, mainly fever and night sweats, the appetite and general nutrition improve, and the larynx, as well as all the other organs of the body, develop greater immunity to infection.

As to the remedial effect of lung immobilization on laryngeal tuberculosis, it is almost the consensus of opinion of the different observers with whom I communicated that a satisfactory collapse exerts a beneficial effect on tuberculosis of the larynx.

Not so long ago the general opinion was that the presence of laryngeal tuberculosis constituted a definite contraindication to the induction of artificial pneumothorax. The present tendency,

however, is to regard the presence of a co-existing laryngeal lesion not as a contraindication but as an added indication for this form of treatment.

The following table will show the remedial effect of lung immobilization on laryngeal tuberculosis:

TABLE NO. 2.

Name	No. Cases of Laryngo-pulmonary Tbc. Treated by Lung Immobilization	No. Cases Arrested or Cured	No. Cases Improved	No. Cases Stationary	No. Cases Unimpro'd
St. Clair Thomson	9	6	0	1	2
Stivelman	5	2	0	3	
Murray	1		1		
Rathbun	2	2	0		
Parfitt	15	8	4	1	2
U. S. Veterans' Bureau	15	3	10		2
Golembe	2	1	1		
McMahon (Loomis)	1	1	0		
Dworetzky	6	3	3		
	56	26	19	5	6

From the above table it will be seen that out of 56 cases of laryngopulmonary tuberculosis treated by lung immobilization, 26 cases had the laryngeal lesion arrested or cured, 19 were improved, 5 were stationary and 6 unimproved.

Case 1.—Case of laryngopulmonary tuberculosis, treated by artificial pneumothorax for six months, also phrenicotomy, with improvement of the larynx. B. F., male, age 49.

Diagnosis: Pulmonary tuberculosis, advanced, active; lesion confined to the right lung; sputum positive; pronounced hoarseness; examination of larynx revealed infiltration of both vocal cords with ulceration. Infiltration of posterior commissure, both ventricular bands and both arytenoids. On May 20, 1929, artificial pneumothorax was instituted with immediate symptomatic improvement, except for cough, which persisted, but to a lesser degree. X-ray examination showed that the right lower lobe failed to yield to compression, and phrenicotomy was performed July 12, 1929, by Dr. Joseph B. Stenbuck, with good result. Examination of larynx November 20, 1929, revealed diffuse congestion of all structures; hyperplasia of posterior commissure

greatly diminished; thickening of cords considerably lessened and voice quite clear.

Case 2.—Case of laryngopulmonary tuberculosis, treated by artificial pneumothorax, reported in 1925, definitely improved. Phrenicotomy was performed in 1929; laryngeal lesion remains arrested.

M. V.—Diagnosis: Pulmonary tuberculosis, advanced, active. History of tuberculosis, about 11 years. X-ray findings, November 10, 1922 showed a heavy infiltration of right upper lobe with evidence of cavitation to a moderate extent. Slight infiltration of middle lobe, merging into peribronchial deposits at base. Left lung apparently clear. The larynx then showed a large infiltrative mass in the posterior commissure, with infiltration of both vocal cords and ulceration of posterior halves. Pronounced edema of both arytenoids. Patient suffered from hoarseness, dysphagia and also dysphonia.

Artificial pneumothorax was instituted in 1923. Under this therapy temperature became normal, all symptoms of toxemia diminished and patient became ambulant. Phrenicotomy was performed October 30, 1929, to aid immobilization of right lung.

Laryngeal findings at present: Very much the same as in 1925, viz., slight thickening at posterior commissure with healed lesion at both vocal processes. Voice is normal and patient has no other symptoms referable to the larynx.

Case 3.—Case of laryngopulmonary tuberculosis treated by artificial pneumothorax and phrenicotomy with arrest of laryngeal lesion. C. K., age 40. Came under my care July 24, 1926.

Initial diagnosis: Pulmonary tuberculosis, moderately advanced, quiescent. Lesion became active and physical examination revealed that the activity was chiefly confined to the right upper lobe, where there was also large cavity formation. Light infiltration of left lower lobe. The larynx showed hyperplasia of posterior commissure with infiltration of both vocal processes.

Diagnosis: Pulmonary tuberculosis, advanced, active. Tuberculous laryngitis, chronic.

Artificial pneumothorax, right side, was induced on October 23, 1929, followed by phrenicotomy on November 3, 1929.

Examination on December 18, 1929, revealed that the right lung was partly compressed; diaphragm considerably elevated and immobilized. There was symptomatic improvement. Laryngeal examination, made May 3, 1930, shows slight thickening of mucous membrane at posterior commissure; both vocal processes completely healed. Voice is clear.

Case 4.—Case of laryngopulmonary tuberculosis, treated by phrenicotomy, with marked improvement in the larynx. M. F., age 43. First came under my care on July 18, 1925.

Diagnosis: Pulmonary tuberculosis, advanced, active, with widespread involvement of the left lung, with definite cavity formation in the upper lobe and extensive pleural thickening. Also signs of early involvement at the right apex. Larynx was found negative. On June 13, 1928, examination of the larynx revealed infiltration of the left ventricular band and also the right vocal cord. There was pronounced hoarseness. Artificial pneumothorax was not attempted on account of pleural thickening. Phrenicotomy was performed by Dr. Stenbuck on May 15, 1929, with almost immediate symptomatic improvement. Cough and expectoration became diminished and temperature normal. Examination of larynx on April 15, 1930, shows that the vocal cords are normal, lesion of the left ventricular band is healed and there is only slight thickening at posterior commissure. Hoarseness has disappeared and voice is entirely normal.

Case 5.—Advanced laryngopulmonary tuberculosis treated by artificial pneumothorax, followed by spontaneous pneumothorax with effusion. Phrenicotomy and extrapleural thoracoplasty left side, in April, 1926. Lesion of the larynx definitely improved.

S. G., age 33. History of tuberculosis, eleven years.

Diagnosis: Pulmonary tuberculosis, far advanced and active; tuberculosis of the larynx, subacute type. Physical findings consisted of widespread infiltration of the left lung with large cavity formation in left upper lobe; also some infiltrative changes in the right lower lobe.

The larynx presented diffuse infiltration of both vocal cords, with considerable thickening of posterior commissure. Patient was very hoarse and at times aphonic.

In October and November, 1925, the patient was treated with artificial pneumothorax. Spontaneous pneumothorax developed, followed in a few days by a large pleural effusion. Patient was kept in bed for several months, and in the course of three or four months fluid was absorbed and there was a well organized hydrothorax. In April, 1926, phrenicotomy, followed by extrapleural thoracoplasty (eleven upper ribs), was performed by Dr. Howard Lilienthal of New York, with gratifying results. The larynx, on repeated examinations between 1926 and 1929, showed pronounced fibrosis of both vocal cords with permanent thickening at the posterior commissure. The other structures were perfectly free. Patient has a good voice of rather low pitch.

Case 6.—Case of laryngopulmonary tuberculosis, treated by artificial pneumothorax followed by partial thoracoplasty. (Resection of upper six ribs); also apicolysis with resection of parts of two ribs anterolaterally. Complete healing of the larynx with the aid of the electric cautery. Case reported by Dr. McMahon, on the staff of Loomis Sanatorium.

F. M. G.—On admission he had a partial pneumothorax of the right lung, which was extensively diseased, and a slight infiltration of the median half of the left lung. Classification: Pulmonary tuberculosis, far advanced, active.

His larynx showed both true cords to be red and swollen, more marked on the left. The left cord was slightly roughened, and the posterior commissure was infiltrated and roughened. A diagnosis of tuberculous laryngitis with ulceration was made. As artificial pneumothorax was incomplete and apparently ineffective, it was discontinued January 26, 1924. Patient was sent to Mt. Sinai Hospital on April 26, 1924, to have thoracoplasty on the right side. At this time he was having some 60 grams of mucopurulent sputum; bacillary content, Gaffky iii. He was running a high fever, maximum between 102 and 103. He was readmitted to Loomis Sanatorium on June 12, 1924, having had a partial thoracoplasty on the right side and also a right phrenicoexeresis.

At this time local symptoms had definitely improved, compared with the time of admission. Cough and expectoration were considerably less, constitutional symptoms were better, the tuberculous laryngitis had not changed. At this time the vocal cords showed thickening and discoloration, especially on the left. Between the insertion of the left vocal cord and posterior commissure there was considerable grayish papillary elevation of the mucosa. His symptoms were soreness and hoarseness and a tickling sensation. X-ray showed moderate fibrotic infiltration with small multiple cavities above the third rib on the right. Light infiltration extending downward and outward from the hilum without much perifocal exudate. A few small peritruncal deposits on the left side. The diaphragm on the right was immobile, and the cut ends of the upper six ribs were clearly demonstrable. He still had considerable bacillary sputum. Patient left the sanatorium on May 28, 1926, to go to New York for consultation on the advisability of further surgical interference. As a result of this, apicolysis was done and sections of two ribs were removed anterolaterally.

He was again readmitted to Loomis Sanatorium on June 10, 1926. This time his laryngitis was considerably improved, but the interarytenoid fold still showed slight erosion and elevation on the left side. Symptoms of occasional hoarseness and tickling sensation, which caused coughing, were still present. On January 4, 1928, because of the persistent erosion at the left of the posterior commissure and symptoms of occasional tickling and hoarseness, it was decided to cauterize it. Two punctures were made, the sloughs of which gradually cleared up, and resulted in marked symptomatic improvement, both subjective and objective. The sputum has been negative for the presence of bacilli from September 11, 1928, up to the time of discharge on October 1, 1929, except for one occasion, in which two bacilli were demonstrated on concentration.

Condition on discharge: Quiescent. Laryngitis, healed.

Laryngeal examination on March 28, 1930, shows hyperplasia of mucous membrane with squaring of the posterior commissure. Also a wrinkling of the mucous membrane on approximation of

the cords. Slight thickening of the left vocal cord but no ulceration.

Case 7.—Case of laryngopulmonary tuberculosis, treated by artificial pneumothorax. Larynx entirely healed. W. H., age 31. Under care of Dr. Harry Golembe.

Diagnosis: Pulmonary tuberculosis, far advanced, with infiltration of upper part of the left lung, with cavity formation. Despite complete bed rest, physical and X-ray examinations disclosed definite advancement of the tuberculous process in the left lung.

Examination of the larynx July 25, 1929, showed a small area of infiltration at the right vocal process, with thickening of the mucous membrane at the posterior commissure. Both ventricular bands congested and slightly edematous. Diagnosis of tuberculous laryngitis, subacute, was made.

After six months of artificial pneumothorax treatment, a complete collapse of the left lung was obtained. Cavities were nearly obliterated. There was an excellent clinical recovery. He now lives under normal conditions; is working full time. A report from Dr. Elmer Funk of Philadelphia, dated April 15, 1930, states: "Patient has a complete artificial pneumothorax. Patient is in good condition generally and is free from local and general symptoms. Larynx is normal."

Case 8.—Case of laryngopulmonary tuberculosis, treated by artificial pneumothorax. Lesion of the larynx definitely improved. S. L., age 38. Patient of Dr. Harry Golembe.

Diagnosis: Pulmonary tuberculosis, advanced and active. Duration of disease, over six years. Clinical course, prior to the induction of pneumothorax, was characterized by constant activity of the lesion, with steadily increasing infiltration in both lungs. Hoarseness of the voice and dryness of the larynx were distressing symptoms.

Examination of the larynx disclosed hyperplasia of posterior commissure, with infiltration of both vocal processes; also infiltration of left vocal cord with ulceration.

Artificial pneumothorax, right side, was induced in December, 1928. In due time the cavities were compressed and the clinical

course was one of continued improvement, as evidenced by gain in weight and disappearance of cough and expectoration. X-ray examination on March 29, 1930, showed right lung well compressed with moderate pleural effusion at the base.

Examination of larynx, May 3, 1930: Voice considerably less husky. Interarytenoid hyperplasia much lessened. Vocal cords, especially the left, show infiltration with superficial ulceration of free border. Laryngeal lesion decidedly improved.

Case 9.—Case of far advanced laryngopulmonary tuberculosis treated by artificial pneumothorax for eight months with decided improvement in both lungs and larynx. Reported in 1925. Larynx remains arrested, although the pulmonary lesion has progressed a great deal.

J. K., age 27. Came under my care July 29, 1921.

Physical examination revealed extensive involvement of right lung, with cavity formation in right upper lobe and infiltration of left apex. There was continuous fever in the afternoon and a great deal of cough and expectoration. Larynx showed infiltration and papillomatous growth at posterior commissure with infiltration of both vocal processes. Patient was very hoarse. Hygienic-dietetic treatment was given for two years without any effect. Artificial pneumothorax was therefore resorted to, and patient showed remarkable improvement. After a few months of treatment a partial collapse of the lung was obtained. Cough and expectoration almost disappeared; temperature became normal.

Examination of the larynx on March 4, 1925, shows all structures to be normal with the exception of very slight thickening at the posterior commissure. Voice is very good and slightly low pitched. After eight months pneumothorax was discontinued on account of adhesions. Examination of larynx October 28, 1929, showed thickening at posterior commissure and vocal processes. Lesion apparently remains healed.

CONCLUSIONS.

1. A satisfactory pneumothorax, by arresting the pulmonary lesion, will almost always prevent the development of laryngeal tuberculosis.

2. A satisfactory collapse of the diseased lung, by either artificial pneumothorax, phrenic avulsion or extrapleural thoracoplasty, usually exerts a beneficial effect on the laryngeal lesion.

7 LAW STREET.

REFERENCE.

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LXXX.

THE USE OF INSULIN IN POSTOPERATIVE MASTOIDITIS WITH PROLONGED DRAINAGE.

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It has been our experience to observe that in any large series of cases of postoperative mastoiditis there are always an appreciable number in which the process of healing is considerably delayed, with persistent discharge exuding from either a fistulous tract or a small cavity in the operative wound or the middle ear. Many of these are seen in cases having no postoperative complications and in which there is no reason to believe that cells have been left unopened or anything but complete evacuation and thorough drainage had been accomplished at the time of the operative procedure.

Why drainage should persist in these cases is not entirely clear. It is our impression that the cause probably lies elsewhere than in the local area and is bound up in some way with the general resistance of the patient to infection and certain fundamental intrinsic powers of healing which at present are not too well understood, but vary in each individual and at different times in the same individual. There is every reason to believe that a person in good health, well nourished and not weakened by a previous long, debilitating disease is apt to display a greater power to heal wounds than another in whom malnutrition, secondary anemia, recent loss of weight and prolonged illness are complicating factors. In this latter group fall many or most of our cases of operative acute mastoiditis. The patient has in the average case been acutely ill for a long period of time, suffers from secondary anemia and is what we call, for want of a better terminology, in a "run-down" condition. Their habits of eating are changed,

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appetites have become poor from prolonged inactivity, and resistance, whatever that may be, is at a low ebb. Such a state we believe is not conducive to the proper healing of wounds, and it is not remarkable that in such cases the process of healing is slow and often incomplete. A vicious cycle is established wherein the debilitated condition prolongs suppuration, and the suppuration aggravates the lowered bodily resistance to infection. These patients do not require a variety of local measures in order for closure to take place, but it is of prime importance that measures be adopted to promote appetite, stimulate metabolism, combat the anemia and improve the general physical condition. In some cases a prolonged but mild degree of acidosis may exist, requiring active therapeutic measures.

In the final analysis it will probably be found that it matters little what methods are used to promote and improve the physical status of the patient, but most of the methods in common use have, in our hands at least, been unsatisfactory.

We have recently adopted use of subcutaneous injections of insulin in a series of cases of delayed healing and prolonged suppuration following mastoidectomy in individuals who are non-diabetic, with results sufficiently gratifying to report in a preliminary fashion. At the present time exact clinical and laboratory data is not collected but will be made the subject of a further and more detailed communication at a later date. The rationale for the use of insulin under these circumstances lies in the repeated observations of Spickard,[1] Short,[2] Marriott,[3] Fischer and Rogatz,[4] Barbour,[5] E. Vogt,[6] Weichman,[7] Moutier,[8] Davidson[9] and Pitfield[10] (who was the first to report such observations), that in malnourished individuals the use of insulin increases appetite and brings about a gain in weight and an improvement in general well being. It is our belief that the return to a normal or increased diet does much to relieve the debilitated state, resulting perhaps in improved tissue resistance and, almost without exception, is followed by more rapid healing than had previously been observed. This is in keeping with Barbour's[5] observations that improvement occurs in the healing of infections following insulin administration. The favorable action of insulin in these cases

may be due to more specific factors than the effect upon appetite and metabolism. For instance, King, Kennaway and Piney[11] have reported certain blood changes following the administration of a single moderate sized dose of insulin. The principal change was an actual increase in the number of lymphocytes and a drop in the number of polymorphonuclear cells. Such factors as these may have a beneficial effect, but exactly what factors are responsible for the clinical improvement noted are merely a matter of conjecture. It is our hope that further and more detailed studies will throw some light upon the fundamental changes involved.

Our method has been to use single daily doses rather than repeated injections. Patients are instructed to present themselves one-half hour before lunch, at which time the insulin is administered. In children the usual initial dose is three units, which is rapidly increased to five or more, according to the reaction observed and the ability of the patient to tolerate larger doses. We believe with J. J. Short[2] that a diet rich in fats rather than carbohydrates is logical, for the appetite created by the use of insulin is soon lost if carbohydrate is taken early in the meal. It must be recognized, however, that a certain amount of carbohydrate is essential, for Woodyat has taught us that in the presence of carbohydrate fat burns, but without it merely smoulders, aggravating acidosis rather than preventing it.

We have used insulin in seven cases so far, each typifying those factors already discussed. We have started its use usually during the sixth or seventh week of convalescence, but considerably later on several occasions, and always in the presence of continued suppuration and an open wound, but chiefly where no progress towards a favorable termination could be observed. In each case, in keeping with the improvement in general health and appetite, there followed a rapid decrease in drainage, new granulations appeared and rapid and complete healing took place.

It must be distinctly understood that this adjuvant in no way replaces painstaking care of the operative wound itself, nor can it be expected to produce favorable results in those cases where the operation has been incomplete and diseased tissue left in situ.

SUMMARY.

1. Many postoperative cases of acute mastoiditis are in a generally debilitated state from prolonged illness and operative procedure.

2. This state of debilitation often prevents the proper healing of wounds.

3. Insulin injections have a decidedly tonic effect upon such cases.

4. Increased appetite, gain in weight and improvement in general health result in increased rapidity of healing.

1915 SPRUCE ST.

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LXXXI.

THE MAXILLARY ANTRA AS FOCI OF INFECTION.*

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The object of this paper is twofold:

1. To present evidence from recent literature and case reports from our records supporting the idea that the maxillary antra commonly act as foci of infection.
2. To stress the importance of careful routine examinations of these regions when searching for the cause of conditions which are generally supposed to be due to such foci.

RECENT LITERATURE.

A consideration of recent literature reveals that quite an advancement has been made within the past few years in our methods for determining diseased conditions of the maxillary antra and that a pronounced tendency toward conservative treatment of all the paranasal sinuses, except possibly the antra, is quite apparent.

The following excerpts and notations taken from a few recent articles give a fair idea of the many and varied diseased conditions which are being attributed to infected sinuses in general and to the maxillary antra in particular:

Kistner, Frank B., Annals of Otology, Rhinology and Laryngology, December, 1929, reports cases covering the following diseased conditions attributable to infected antra:

1. Myocarditis.
2. Chronic bronchitis and perennial asthma.
3. Retrobulbar neuritis.
4. Chorea of three years' standing in a child aged 9. Removal of tonsils and adenoids had done no good. Cured by antrum operation.

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*Read before the Northwest Texas Medical Association, Wichita Falls, Texas, September 9, 1930.

5. Chronic bronchitis in a girl aged 14. Cured by sinus exenteration.

Houser, Karl Musser, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, March, 1929:

1. Vertigo. Well for eight months after window was made in one antrum.

Ernser, Matthew S., ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, March, 1929:

1. Headache, cough, profuse nasal discharge, eye disturbance, anemia.

2. Headache, dizziness, constant cold in nose and discharge, foul breath.

3. "In a group of diabetics with hyperglycemia we found that the blood sugar could not be reduced until the maxillary antra were operated upon, thus removing the source of infection (Caldwell-Luc performed)."

Emerson, Francis P., ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, March, 1928:

1. "One class of cases is simply inconvenienced from what seem to be frequent colds. A second class suffers from headaches, neuralgia, fatigue or a bad feeling head; while a third class is invalids from a *blood stream infection* of some distant organ."

2. Asthma of twenty years standing. But one attack in twenty-two months following Caldwell-Luc operation.

3. Constant colds, specks before the eyes, with repeated attacks of myositis.

4. Headaches and trifacial neuralgia. Nine months after double Caldwell-Luc operations, no pain.

5. Subacute conjunctivitis and head pains.

Carmody and Green, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, September, 1929: "We take the stand that without infection in the sinuses or without pathologic membranes, our irritants, such as dust, pollens, bacteria, foods and emanations of all sorts, etc., do not produce the so called allergic coryza or hay fever."

Kistner, Frank B., ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, September, 1929:

1. Diseases of the rheumatic group. Report of a case of subacute polyarthritis of nine months' standing due to nonsuppurative infection of the antra. Both antra cultured streptococci with a specificity for the joints of rabbits.

2. "So far we have found definite foci of infection in every tic case we have examined."

3. "From a clinical standpoint it is often the nonsuppurative that do the most damage."

4. "Sinus infection has been a factor in a number of troublesome disease conditions not so well defined, as in the following: Secondary anemia with chronic cholecystitis and chronic nonpurulent sinusitis; tachycardia with dyspnea and fatigue of unknown origin; infectious atheromatous type of cardiovascular disease with cardiac failure."

5. "I am firmly convinced that the infection in chronic sinuses is as great if not a greater source of trouble than the chronic tonsils, and our failure to recognize it is responsible for our failure to relieve many of our patients suffering from focal infections."

6. "From the clinical standpoint, practically any disease which can be produced by focal infection anywhere can be produced by sinusitis."

Mithoefer, Wm., Larygoscope, January, 1929:

1. "Careful examination of many patients, however, has convinced us that this cavity is many times mildly or markedly involved, and that the pathological changes in the antrum have very often remained unrecognized because of the lack of symptoms referable to the nose."

2. "The marked involvement of the general health of many of our patients, and the rapid gain in weight following the removal of pathological tissue from an unsuspected antrum, is proof, in our estimation, that chronic toxemia from this cavity is very often a sufficient reason for the patient's state of ill health. It has impressed us also with the pertinent fact that a toxemia may be caused as readily by a low grade infection, without demonstrable pus within a bony cavity, as it is caused by a purulent infection."

"Case 5: Mrs. P. M., aged 60 years, has suffered for the past five years with a tic douloureux on the right side. It was decided to do an immediate radical antrum operation.

Comment: The patient has been entirely relieved of the tic douloureux since the operation."

"Case 7: Mrs. N. W,. age 40 years, consulted us on March 3, 1928, with a history of having had an incessant cough for the past four years.

Comment: Almost immediately following the last operation, there was complete disappearance of the cough."

Radical ethmoid and antrum operations were done in this case.

PERSONAL CASE REPORTS.

Case 1. Persistent Corneal Ulcer. Mrs. W. R. H., age 44. Healthy housewife and mother of several healthy children, developed an abscess of the right lower eyelid near the outer canthus. Following this a large corneal ulcer developed which at first was shallow, but later became deeper and very painful. The lid abscess healed in due time but the corneal ulcer resisted all ordinary methods of therapy, including several milk injections, though it would at times look as if it were going to heal. Patient's mouth had been edentulous for some years, tonsils were of fair color and contained some débris, and there was no evidence of sinus involvement according to interpretations then (1925) popular for making such diagnosis, save the right antrum was a slight bit darker than left on transillumination and this was not corroborated by X-ray pictures. After about three weeks, all suddenly hypopyon appeared and the tonsils which had been firm and of fair appearance became mushy and fairly oozed pus. Feeling that, after all, the tonsils had been worse than I had thought them to be and that I had at last located the hidden focus, I removed them. During the following two weeks I was away from my office and a confrere attended my patients. On my return I was dismayed to find that this corneal ulcer, while improved, still persisted and still pained. As a last resort, I decided to irrigate the right antrum, even though evidence of pathology was so meager. The first irrigation through needle puncture brought away much foul smelling pus. Ten days later

patient was discharged with the ulcer entirely healed and the return flow from the antrum clear on the fifth irrigation.

Comment: Manifestly the antrum was the original focus of infection all the while, which furnished the focus leading to the abscess of the eyelid, the persistence of the corneal ulcer and accounted for the apparently sudden appearance of so much pus in both tonsils.

Case 2. Facial Paralysis. Mr. M. K., age 33, some eight weeks previously had developed right sided facial paralysis since which time had been examined by two competent groups of clinicians with negative findings. According to patient's statement his sinuses had been passed over in both instances as negative without transillumination, X-ray pictures or the use of the nasopharyngoscope.

Examination: X-ray picture was negative for all sinuses. Transillumination showed left antrum distinctly darker than right. Needle puncture and irrigation of left antrum revealed slightly cloudy fluid the first time, followed by much mucopus on second and third irrigations. Temporary windows were made in both antrums, the right showing suspicious return flow on the first irrigation, following which both promptly cleared. The facial paralysis disappeared completely within the following few months without further treatment of any kind.

Case 3. Tic Douloureux with lower half headache. Mrs. J. G. B., age 40 years, had suffered on two previous occasions with typical right lower half headache, relief having been obtained both times through applications of cocain and silver nitrate to the right sphenopalatine ganglion. Something over a year following the second of these, the same dull pain began in the same region and in addition she shortly developed typical tic douloureux symptoms affecting the right side of the face. These symptoms persisted with increasing intensity for some weeks prior to her return to me June 22, 1929, during which time general physical and otolaryngological examinations were pronounced negative.

Examination: Cocain applied to region of right sphenopalatine ganglion would give temporary relief from all symptoms. X-ray pictures of sinuses were negative. On transillumination right

antrum was slight bit darker than left. There was some engorgement of the right inferior turbinate. On these findings, together with the negative general physical, it was decided to do needle puncture of the right antrum.

Treatment: Irrigation through needle puncture gave a perfectly negative return flow the first time, while the second was slightly cloudy and with small clumps. Eight irrigations in all, given within two weeks, gave a clear return flow again at the end of the time with a decided abatement of the tic symptoms. A report from this patient who lives in a distant city two months after returning home was to the effect that she had been absolutely free from symptoms for the past month and a report ten months later was that she was still symptom free.

Case 4. Mrs. F. M. B., age 54. Chief complaint was that for two years she had suffered almost constantly with a dull pain affecting the left forearm and hand with a sense of numbness in the same region. Frequently the pain was so intense she could not sleep nights. In addition there were attacks now and then of an equally severe pain affecting the left sciatic nerve.

Routine examination resulted in suspicion of infection in both antra which was confirmed by needle puncture. Windows in both antra resulted in complete relief from all symptoms within about three months and patient is still symptom free eighteen months later.

Case 5. Acute Arthritis. Dr. S. S. G. gave a history of having had a previous attack affecting right knee which yielded to ordinary measures. Present attack affecting right ankle joint, however, would not yield to anything thus far tried. No focus of infection had been located until on routine examination suspicion as to the right antrum was developed. Needle puncture confirmed the suspicion of infection and repeated irrigations over a period of two weeks resulted in clearing antrum and at the same time completely relieving the arthritis.

Comment: This patient was getting about on crutches when the trouble was first located and drove his car home within a few weeks thereafter. He is still perfectly free from symptoms seventeen months later.

Case 5. Symptoms Simulating Tuberculosis and a distracting sphenopalatine ganglion pain. Miss G., age 45, came with a history of having had a profuse nasal dropping, annoying productive cough and typical sphenopalatine ganglion pain for two or three years. Her general appearance was that of a well advanced case of pulmonary tuberculosis and had been advised at one clinic to go to a tubercular sanitarium, though tubercle bacilli could not be demonstrated. There was no history suggestive of tuberculosis. Routine examination revealed bilateral infected antra and double Caldwell-Luc operation followed by a stormy convalescence resulted in complete restoration. She is still well one year after operation.

COMMENT.

From my records covering a practice of over thirty years I could multiply these cases by the hundreds, but I deem these sufficient to impress the point which I am trying to make and that is that infected antra can be and often are responsible for these different disease conditions and it is certainly most important that we make it our habit to routinely investigate the antra in every case that comes under our care. I do not mean by this that we should do needle puncture and irrigate every case, but we can take the time to take a careful history, transilluminate, note the condition of inferior turbinates and use the nasopharyngoscope. If done routinely these will require but little time and will many times lead to detection of latent antrum infection which otherwise would not be suspected.

DIAGNOSIS.

While the diagnosis of antral infection is without the scope of this discussion, I want to say that patience and skill are often required in order to arrive at a fair conclusion as to whether an antrum is infected. Sometimes it comes to a matter of the elimination of all other possible foci coupled with questionable antral findings; but patience and perseverance have frequently led me to a conclusion which ended happily for both the patient and myself. In allergic patients it is sometimes hard for me to satisfy myself as to whether the allergy, through blocking, has preceded the

infection or whether the infection has been responsible for the allergic symptoms. At any rate, allergy should always be kept in mind.

SUMMARY AND CONCLUSIONS.

Evidence as gleaned from widely different sources in recent literature and personal experience of over thirty years indicates that any disease condition which may arise from a focus of infection anywhere in the body can and often does arise from diseased maxillary antra. This being true, the antra should be routinely investigated when searching for foci of infection. Unless a routine is established and adhered to, many infected antra will be overlooked.

I would not conclude without mentioning a recent article by Anderson (Anderson, C. M., Jour. A. M. A., June 14, 1930, p. 1889), of Rochester, Minnesota. I mention this article only to express my hearty disagreement with its general import. Under "Summary and Conclusions," among other things, Anderson says that "Suppurative sinusitis may be a focus of infection on rare occasions." I do not feel that this view, by any means, expresses the consensus of opinion of present-day otolaryngologists.

LXXXII.

THROMBOPHLEBITIS OF THE INTERNAL JUGULAR VEIN AS A COMPLICATION OF TONSILLITIS.*

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AND

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As a fatal complication of tonsillitis, thrombophlebitis of the internal jugular vein probably takes first rank. Although tonsillitis is one of our oldest and most common diseases, yet the acute and fatal complications arising from this disease have not been thoroughly studied. The overwhelming septicemia and the meningitis, which so rapidly develop in the terminal course of the disease, so overshadow the primary focus that the complete pathology has been almost entirely overlooked.

In going over a series of fatal cases of thrombophlebitis of the cavernous sinus, we were surprised to discover in nearly every case a complete clinical picture of a thrombophlebitis of the internal jugular vein. It was not until 1928 that we recognized the direct relation of the septicemia with involvement of the internal jugular vein to that of tonsillitis. An additional factor in the failure to recognize the primary phlebitis is that the tonsillitis is usually mild, that it is the first attack and promptly clears up with the onset of the septicemia. This, we believe, readily explains why our investigations have been misdirected. The involvement of the internal jugular vein is practically always unilateral. It causes very slight local reaction, practically no pain, there is moderate tenderness over the course of the vein and hardly enough swelling to recognize. As there is no impairment

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of function or motion, it is little wonder that the disease has been so seldom recognized. Altogether, eleven cases of thrombophlebitis of the internal jugular vein have come under our observation, but, unfortunately, we have recognized only eight as a distinct clinical entity.

Two days prior to our first patient's death a tentative, and so far as we knew, original diagnosis of thrombophlebitis of the right internal jugular vein, complicating tonsillitis, was made. On later and more thorough investigation we found that Waldapfel[1] et al. had four years previously investigated and reported upon this condition. In 1917, Goodman[2] reported one case in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, upon whom he had resected the internal jugular vein for a phlebitis complicating tonsillitis. The patient recovered.

The following cases are a few of the typical ones which show the course of the disease:

CASE REPORTS.

Case 1.—On the 21st day of March, 1928, one of us (O. J. D.) was called to see an eighteen-year-old student nurse on account of a sore throat. She had been in training about a year, had always been well and never had a sore throat.

There was a thin, gray, streptococcic-like membrane over both tonsils, which were large and red but not markedly swollen. There was a moderate subtonsillar adenitis. She did not seem to be very sick, although she gave a history of a severe chill at the onset of this illness two days before, and another chill twenty-four hours later. Cultures taken from the membrane showed streptococci and no Klebs-Löffler bacilli. On account of her chills and fever of 103° F., she was transferred to the hospital.

On the 24th of March she asked to go back on duty. Her temperature at that time was 100° F. and she had no complaints, except that she was a little tender on the left side of her neck which was slightly swollen. This swelling, however, seemed to be deep. The superficial tissues were not inflamed, and there was no edema. The glands were not swollen, except directly beneath the tonsil. The membrane over the tonsils, at this time,

had completely disappeared. That afternoon she had a severe chill lasting twenty minutes, followed by a temperature rise of 104° F. and 18,000 leucocytes with 90 per cent polymorphonuclears. Blood cultures taken at this time were negative. She also developed, following the chill, some pain on deep inspiration just below the lower end of the sternum. The respiratory rate was moderately increased, and we thought she might have a complicating pneumonia. Physical findings in her chest, however, were negative. There were six to eight pus cells in the urine per high power field.

The following morning, twelve hours later, she had another severe chill with a temperature rise of 106° F., and began to show the yellowish, faded out color of a septicemia. The chills continued without intermission, every twelve hours, with the temperature going as high as 107.6° F. The swelling on the left side of the neck increased, although it was always a deep seated swelling, with no superficial pointing or redness of acute inflammation. She was extremely tender over the entire course of the left internal jugular vein.

At this time we made a diagnosis of a thrombophlebitis of the left internal jugular vein following tonsillitis, thinking the same process had taken place that would have happened had the infection originally been in the ear, except that the infection was coming from within outward instead of from without in. Aside from the chills and high fever, her only complaint was pain in the lower right chest. A pleural friction rub could be heard and her respiratory excursion was diminished. Nine days following the onset of her sore throat she died from the exhaustion of her septicemia.

At the autopsy there was swelling of the neck on the left side and a faint lemon tinged skin. Otherwise nothing of particular interest was noted on external inspection. The abdominal organs were essentially negative except for some cloudy swelling of the liver and kidneys, and a rather soft, toxic spleen. In the chest both lungs were riddled with varying sized, poorly outlined, early abscesses, peripherally distributed, varying in size from 1 to 6 cm. They were covered on the pleural surface with a thick,

fibrinopurulent exudate. Histologic examination revealed early embolic abscesses without complete destruction of the pulmonary parenchyma.

The left internal jugular vein presented a very interesting picture, both grossly and microscopically (Fig. 1). This vein, which was filled with friable, greenish-black thrombotic material, was dissected loose from below up to the angle of the jaw. The larger ramifications were also filled with septic thrombotic masses. There was considerable surrounding inflammatory edema, and, histologically, the perijugular lymphatics (Fig. 2) as well as the lumen of the vein, contained masses of pus cells and necrotic débris in which streptococci were found. The tonsils showed a subsiding inflammatory reaction, no acute process being present.

In the cranial vault the petrous portion of the temporal bone was chiseled away and the jugular bulb and lateral sinus were found filled with the same type of necrotic thrombi as that in the jugular vein below.

Smears and cultures from the abscesses showed a streptococcus. The other viscera presented nothing of particular interest.

Case 2.—Mrs. S., aged thirty years, was admitted to Isolation Hospital on September 1, 1928, with a temperature of 106.2° F., a sore throat, was semidelirious, and with a very rigid neck. Her admission diagnosis was meningitis. The following day she gave us this brief history: She had been ill about ten days with an acute sore throat, the first that she had ever had. On the fourth day her throat was so sore that she could not swallow, and a doctor was called who lanced her throat on both sides. Following this she bled a great deal but saw no pus. She had chills and became much worse after this, developing a severe headache, her neck becoming sore and rigid, forcing her to hold her head to one side.

On physical examination the patient appeared to be suffering from a severe form of meningitis and was very sick. She had a yellowish, septiclike jaundice. Her eyes reacted normally, and, while it was an extreme effort for her to talk, yet she answered questions clearly and accurately. The ear examination was normal. Her teeth had all been extracted several years prior, and

the tongue, which was heavily coated, deviated sharply to the left. Her entire throat was inflamed and edematous, involving the tonsils, peritonsillar space, uvula and pharyngeal wall. There was a serosanguinous discharge from a small incision in the left peritonsillar space. There was marked tenderness over the course of the left internal jugular vein, which was not palpable. The spinal puncture showed 2,400 cells but without bacteria. Her white blood count was 10,700, urine normal, and the culture from the throat showed many cocci in chains.

COMMENT.

The etiology of this very obvious meningitis was, primarily, a streptococcic tonsillitis with resulting thrombophlebitis of the internal jugular vein with extension upward to the meninges. The jaundice was part of the streptococcic septicemia. The onset, course and termination were almost identical with that of case number one, except that this patient had incisions into the peritonsillar spaces on the fourth day of her acute tonsillitis. It is difficult to prove, but we believe that surgical trauma plays an important part in the etiology of thrombophlebitis of the jugular vein.

NECROPSY FINDINGS.

The lungs showed areas of septic infarction, some of them having undergone partial autolysis with beginning abscess formation. The neck, on dissection, showed some purulent cellulitis in the deep tissues, and the internal jugular vein showed septic thrombi of a gray, purulent character, which, high up in the neck near the angle of the jaw, were adherent to the vessel intima. The brain showed numerous hemorrhages extending up along the base in the dura, and a rather marked purulent basilar meningitis. The left jugular vein and bulb were filled with solid septic thrombi. The right jugular bulb, however, showed no evidence of infection. (Fig. 3.)

Case 3.—Twenty-two days prior to her death, W. W., a 19-year-old girl, developed an acute sore throat. It was worse on the fourth day and she called her doctor, who told her that the

left tonsil was normal but that there was a marked swelling of the right side of the neck over the course of the internal jugular vein and a bulging of the right peritonsillar space. This peritonsillar swelling was incised and some pus was drained out. She promptly started to have severe chills, as many as three and four a day, which lasted about one week. Seven days prior to her death, and fourteen days from the onset of her sore throat, the right eye protruded to such an extent that the lid would not close over it. There was a marked palpable pulsation of this eye, which receded two days later, about as suddenly as it occurred. That night she became delirious and the next morning developed acute, severe pain in her chest. At no time was the left eye involved. She died without regaining consciousness two days later. There was a history of one attack of acute tonsillitis two years prior, and she had had a carbuncle on the left forearm six months previous to her death.

COMMENT.

Unfortunately, this patient was not confined in a hospital during her illness and no laboratory work was done. Again etiologic importance of trauma makes it a debatable point. If it did not have any etiologic significance there was probably enough pathology in the throat to produce a thrombophlebitis of the adjacent veins. The primary involvement of the right cavernous sinus, with recession of the eye, is an interesting finding. It is also important to note the rapid and fatal complications which developed immediately after this recession. That the opposite half of the cavernous sinus was not involved is due to the fact that the septic thrombus became purulent, and the pressure from the venous stasis washed out this purulent débris into the jugular vein, and the lungs were immediately filled with septic infarcts. This pathologic process is identical with one of the three cases which came under our observation, but was unrecognized at first as a thrombophlebitis of the internal jugular vein. It is obvious that no surgical intervention was possible. The previous history of acute tonsillitis is out of line with most of the other cases, and the carbuncle on the arm probably had no direct bearing on the fatal complication.

NECROPSY FINDINGS.

On external inspection at autopsy there was no gross swelling of the neck and neither one of the eyes protruded. The pleural surfaces of the lungs were covered with a thick, fibrinopurulent exudate in which fibrin was abundant and of yellow color. Both lungs were riddled with embolic abscesses and areas of embolic pneumonia. Histologically, the thrombi in the heart and lungs showed a rather unusual thrombus formation, the large majority of the cells composing them being mononuclear. The abscess cavities in the lungs had undergone considerable autolysis and contained many masses of bacteria.

The internal jugular vein showed marked inflammatory fibrotic thickening of the wall. The lumen of the vein was lined with necrotic, fibrinopurulent, granular exudate in which masses of bacteria were seen. In the surrounding vessels, particularly in the lymphatics, small purulent thrombi were seen. The pituitary gland showed suppuration over the surface and necrotic thrombosis in the vascular channels surrounding it, but there was no actual invasion by leucocytes. The cavernous sinus was plugged with the same type of thrombi as were found in the heart and lungs.

Case 4.—A thirteen-year-old schoolgirl, B. R., was admitted to St. Luke's Hospital on May 7, 1929, on account of difficult breathing, a swollen right neck and intermittent bleeding from her throat. Three weeks before she had had her first attack of severe tonsillitis. Three days from the onset, on account of difficulty in breathing, her family physician was called, and he lanced the right side of her throat on account of what he described as an abscess. The patient obtained no relief, and three days later began bleeding quite freely from the incision. At this time the right side of her neck began to swell, and the parents noticed that she had difficulty in closing her left eye; the left side of her face was flat and did not wrinkle; and her left arm and leg were partially paralyzed. Her temperature upon admission was 102° F., and she had 22,100 leucocytes, and three to eight pus cells in her urine. The mass in the right side of her throat was opened through the old incision with a hemostat but no pus was evacuated. This mass was found to be a hematoma.

Neurologic consultants suggested the probability of brain abscess in the right temporoparietal region, but they advised waiting for exploration. Her speech at this time was very high pitched and quite unnatural. She slept fairly well, took only a moderate amount of fluid, and would always cry out when she made an effort to move either the left arm or leg. There was a marked improvement, however, in this paralysis. There was a papilledema on the right side. One week following her admission her temperature became normal; she began to show marked improvement in the mental disturbance, and the paralysis of the left leg cleared up entirely. She could move her left arm with less effort. After one week of normal temperature—that is, fourteen days from her hospital admission—at 7:15 o'clock in the evening she suddenly and without warning started to bleed from the old peritonsillar incision. The intern was unable to control this bleeding, and when we saw her an hour and a half later she had lost over a pint of blood. Her blood pressure had dropped so low that she had stopped bleeding, and the nurse, having given her two one-sixth grains of morphin hypodermically without our knowledge, the child was sleeping soundly. We noticed that she had at the time a peculiar respiratory rattle. Her pulse rate was 136. Her mother, father and an uncle were typed for donors and a blood transfusion was contemplated. Due to the fact that she was sleeping and had apparently ceased bleeding, this blood transfusion was postponed. At 2:45 o'clock the following morning the patient was dead.

COMMENT.

This patient presents an unusual number of freak complications. First, a hematoma of the peritonsillar space; second, a left hemiplegia with partial spontaneous recovery; third, repeated uncontrollable and, eventually, fatal hemorrhage from the right peritonsillar space; fourth, a septicemia, and fifth and finally, aspiration hemorrhage into the lungs. As shown by autopsy, all of these complications might be explained by one pathologic lesion—i. e., thrombophlebitis of the right internal jugular vein. This might have produced a venous stasis of the peritonsillar veins which, in turn, caused the fatal hemorrhage. The encephalomalacia is not so easily explained, since it was apparently not infected.

NECROPSY FINDINGS.

On external inspection there was considerable hemorrhage of a frothy character coming from the mouth, and the right side of the neck was markedly swollen. There was a large clot, about three and one-half centimeters in diameter, which was pulled loose from the mucous membrane behind the pharynx. The tongue was deeply injected.

On histologic examination, sections taken through the area of the brain where the acute encephalomalacia was seen, showed acute vascular hyperemia and a typical area of encephalomalacia with breaking down of the brain substance. Section through the neck in the region of the vagus nerve showed a rather marked leucocytic reaction and hemorrhage about the nerve. The tonsil presented an acute hyperplastic type of lymphoid proliferation. Section through the large thrombotic mass which was found in the neck showed a typical laminated thrombus, markedly infiltrated with pus cells.

Case 5.—On the 13th day of May, 1928, we were called to see a twenty-eight-year-old woman, R. L., on account of a painful, stiff neck. She gave the following history: One week before, following a moderate sore throat, her temperature rose to 103° F., and her right neck became so sore and painful that she could not turn her head. Her family physician was called and heat was applied. The swelling and pain became progressively worse, and when we first saw her there was a red, swollen, tender mass extending from the right mastoid tip to the clavicle. It was difficult for her to open her mouth, and some swelling extended forward over the right angle of the jaw. She was brought to the hospital, and for five days her temperature fluctuated between 99° F. and 103.6° F. She had no more chills; her blood findings were 80 per cent hemoglobin, 4,420,000 red blood cells, 16,800 leucocytes with 81 per cent polynuclear cells. Urine examination showed 10 to 20 pus cells per high power field. Blood Wassermann was negative. Her previous history was essentially negative. She had had considerable tonsillitis when a child, she had a tonsillotomy at fifteen years of age, and one year later the remainder of her tonsils were removed. At the time of her hospital admittance

her sore throat had entirely disappeared. There were no bad teeth and there was no evidence of the initial infectious lesion. After six days this mass in the right side of the neck localized and pus was aspirated. It had, however, extended below the clavicle (Fig. 4), and the mass, the size of one-half of a small grapefruit, was incised under local anesthesia. Six or eight ounces of thick yellow pus was drained off and an area the size of one's hand had undermined the skin and deep subcutaneous tissues. Another incision was made midway between the mastoid tip and the sternum attachment, and about three ounces of pus were obtained from this abscess. The clavicle interrupted the drainage of this area through the lower incision. Hot fomentations were applied and her postoperative convalescence was uneventful, with a normal temperature for ten days, when suddenly she began to bleed from this incision and the entire undermined cavity filled with a hematoma. It was difficult to determine just what vessels were involved in this hemorrhage without a rather mutilating operation, and as there were only two points from which the blood could escape (the upper and lower incision), these small wounds were sealed over with layers of cotton and collodion and firm pressure bandage was applied to the entire area. This controlled the bleeding and no further trouble was experienced for another week, when this hematoma became infected, her temperature reached 102° F. for one day, which quickly receded as the infected clot began to drain from the lower incision. Following the hemorrhage her hemoglobin fell to 52 per cent; she had 2,220,000 red blood cells and 13,600 leucocytes. Intra- and extracellular diplococci, which were gram positive, were found in the pus. She was dismissed from the hospital after six weeks and has had no further trouble. (Fig. 5.)

COMMENT.

Due to the fact that this patient recovered from this rather unusual infection, the complete pathology of this lesion is, of course, speculative. Her history, however, was so typical of our fatal cases that we feel that she developed a septic thrombophlebitis of her right internal jugular vein and adjacent superficial

veins; that the process was localized, walled off, and broke down so completely that a general septicemia did not occur. That it was a rather vigorous type of infection was shown by the widespread area which was involved. The comparatively superficial involvement was, no doubt, the factor which saved the life of the patient.

Case 6.—D. G. This is one of the most interesting and instructive cases that has come under our observation, for the following reasons:

First, because this is our only patient who has recovered from a proven thrombophlebitis of the internal jugular vein, and second, because we were able to follow the entire clinical course of the disease which paralleled the entire course of the fatal cases so closely.

This seventeen-year-old girl had always been well, and had never had a sore throat until she attended a New Year's dance in 1930. She became chilled on the way home from the dance and developed a sore throat. She had a fever of about 103° F. and profuse sweats for three days, and at 4 o'clock in the morning of January 3rd she had a severe chill, another at noon that day and one again that evening. Following these chills, her temperature rose to 105° F. The throat remained sore and she had pain down the right side of her neck, at which time she noticed a swelling over the right side of her face and neck. Her tonsillitis, which had begun on the left side, got much better and she was fairly comfortable, except for the high fever, which continued up to the time of her hospital admission. Her attending physician, Dr. Waller Hook, suggested that she had developed a thrombophlebitis of the internal jugular vein, as a complication of the tonsillitis. About the only finding which indicated this complication was slight tenderness over the course of the right internal jugular vein.

Based upon her history, the acute septicemia and these rather vague physical findings, we decided to resect the right internal jugular vein. This was done under ether anesthesia, and the vein was found to be completely thrombosed. Microscopic sections from the vein wall removed at operation showed adherent thrombotic material which was undergoing organization

with fibroblasts and capillaries growing into the thrombus from the subendothelial stroma. The endothelium was missing. About the vessel the connective tissue and muscle showed considerable replacement fibrosis and mononuclear infiltration. Due to the marked extension of the thrombus, normal vessel wall was not reached, and the course of the septicemia was unaltered. She was given a transfusion of 500 cc. of blood and her recovery seemed very doubtful. Her septicemia continued, the pain in her right chest disappeared but persisted in her left chest. She lost weight rapidly but continued to eat and took fluids very freely.

Although the throat and neck infection entirely cleared up, she developed multiple lung abscesses and a subsequent empyema of her left chest. A rib resection was done. This patient has completely recovered, regained her normal weight and suffered no ill effects from this unusually severe illness.

COMMENT.

Earlier diagnosis and more prompt surgical intervention might have prevented the pulmonary complication. However, as the patient recovered, it is unfair to state just what changes should have been made in the management of this case. Except for the prevention of a retrograde thrombosis, we doubt if the resection of the jugular vein in this particular case was of any benefit, since the thrombus had already undergone early organization and the vein was obliterated. The pulmonary complication had already taken place at the time of the resection, and the thrombophlebitis had extended too far down into the chest to be reached surgically. The fact that the patient continued to assimilate large quantities of fluid throughout the course of her illness was, we believe, a great factor in her recovery.

SUMMARY.

Thrombophlebitis of the internal jugular vein may develop as an extension of an acute and overwhelming infection which has its origin in the tonsil. While the rich vascular supply of the tonsil usually prevents this complication, yet, when the infection breaks through into the vein, an overwhelming and fatal septicemia usually occurs. Trauma to the inflamed tonsillar or peri-

tonsillar tissues predisposes to this complication. In most cases the phlebitis occurs as a complication of the primary attack of tonsillitis, which would indicate a lack of local resistance and a lowered immunity to the infecting organism. We believe that the lymphatics play only a minor part, that perivascular abscess formation rarely occurs and that the thrombophlebitis is similar in every way to septic thrombophlebitis in other veins of the body, as, for example, the sigmoid sinus in mastoiditis. We also believe that this condition has existed for as long as we have had tonsillitis and that, if recognized early, it may be amenable to surgical intervention. The resection of the infected vein and removal of the thrombus is always indicated when a positive diagnosis can be made early enough to warrant intervention. All forms of surgical trauma to the acutely inflamed tonsil or peritonsillar spaces should be avoided. The chief progress that we shall make in the recognition of this disease will come by our knowledge gained in complete necropsy studies.

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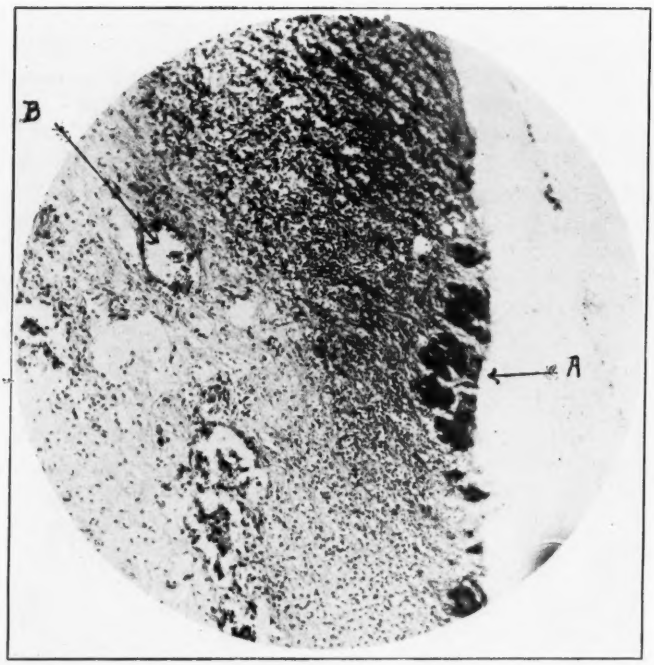

Fig. 1. Section through the wall of internal jugular vein. Mag. x 125.
(a) Septic thrombus in the lumen.
(b) Dilated lymphatics in the wall containing pus.

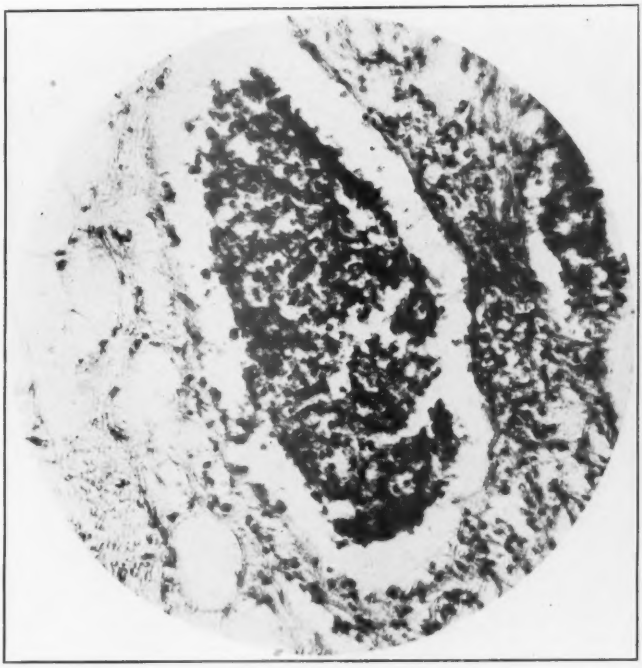

Fig. 2. Perijugular lymphatic channel filled with necrotic pus cells. Mg. 500 X.

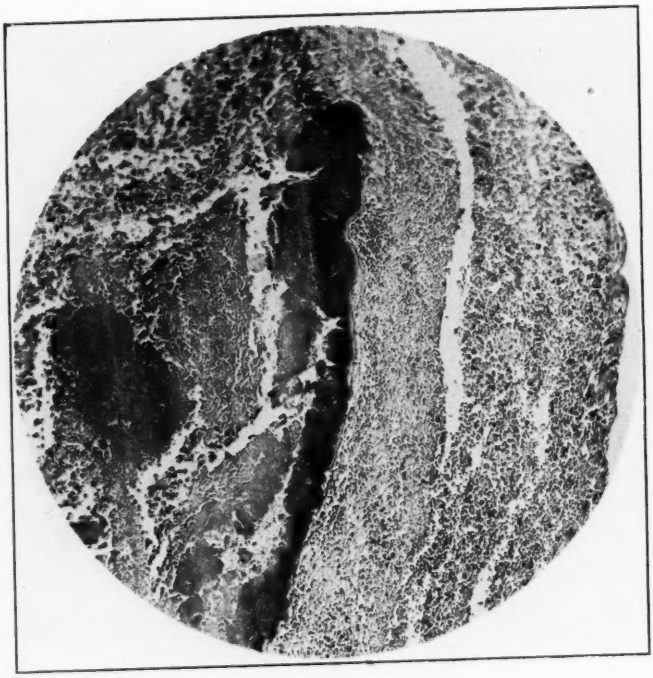

Fig. 3. Jugular vein filled with autolytic suppurative thrombotic material. Mg. 125 X.

Fig. 4. Photograph taken six days following onset of acute tonsillitis, showing the abscess extending from the angle of the jaw below the sternum. The external jugular vein had thrombosed and broken down.

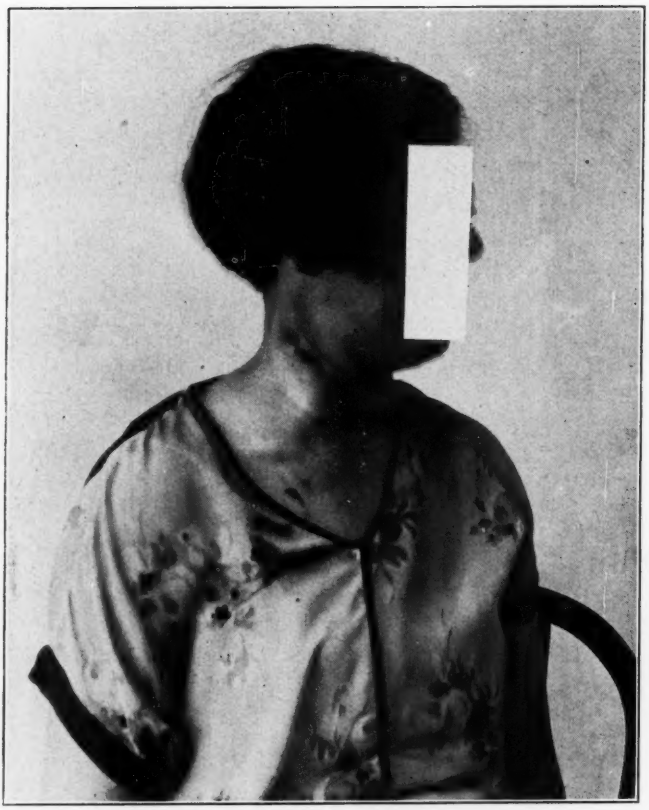

Fig. 5. Six weeks postoperative showing the scars where the abscess was drained.

LXXXIII.

BACILLUS PROTEUS VULGARIS INFECTION IN SECONDARY MASTOIDITIS, FOLLOWING TONSILLECTOMY, AND IN RADIUM IMPLANTATION IN TONSILS.

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The rôle of the bacillus proteus vulgaris in surgery is as a rule a terminal one. These organisms have their normal habitat in the gastrointestinal tract and may spread or be carried to any other part of the body. The following three cases demonstrate this type of infection in the ear and the pharyngeal tract.

Case 1.—Boy, E. J., 18 years of age, referred for swelling and pain in the region of an old mastoid wound. He had a severe rhinitis for a few days, with pain in the right ear, and an increase of the chronic discharge which followed a previous mastoidectomy. After several sleepless nights a swelling and redness appeared over the old scar.

I found a fluctuating red area covering the scar of a previous mastoidectomy, with quite a bulge at one point. On further questioning, I elicited the following interesting information as to the operation. At the first dressing, there was a very severe hemorrhage, requiring his removal to the operating room, after which he was critically ill for several weeks, and ran a high temperature. This sounded like a sinus hemorrhage, and in opening the mastoid cavity the incision was made away from the old scar. There was a gush of dirty, gray, foul-smelling pus as soon as my knife went through the superficial skin. This opening when enlarged presented the usual findings in a secondary mastoid operation, with layers of new bone formation, and the complete exposure of the sinus which was adherent in part to the old scar and covered by dirty gray patches of granulation tissue. The sinus was pulsating and was consequently not disturbed. The rest of the cavity was cleaned out thoroughly and the antrum drained.

Culture of the pus showed the bacillus proteus vulgaris.

Wet dressings of Dakin's solution were used and changed daily because of the powerful stench. After a few days a change was made to permanganate solution 1/5,000. The patient did well up to about the seventh day and then began to vomit, being unable to retain anything. The temperature, which had been flat for about three days, now went up to 101. He became listless, extremely weak and seemed to have lost a good deal of weight. He was unable and unwilling to get up or take any interest in his surroundings or in those who visited him. He just wanted to stay in bed and be left alone.

Blood count, 12,500, with 85 per cent polymorphonuclears. Urine negative. Blood culture negative.

There were no cerebral signs, no nystagmus, no reflex changes. Careful dieting, stimulating medication and constant, painstaking dressings gradually brought him around, and after a three weeks' stay in the hospital he was removed to his home, in the hope that the change of atmosphere would do him some good. After a week at home he again began to vomit and rapidly lost ground, becoming extremely toxic and losing about thirty-five pounds in weight. He was put to bed, and under the careful guidance of his family physician, Dr. J. G., started to regain his weight and strength. The wound at this time was closing over, and the odor also was gradually becoming less offensive, until, at about the end of the seventh week, it was practically gone. The patient, at the present time, twelve weeks later, is up and about, has regained his weight and has just a small amount of discharge from a perforation in the middle ear. The culture still shows the bacillus proteus, which has apparently changed to another type or strain.

Case 2.—Woman, age 23, in excellent physical condition, except for diseased tonsils, which have given her frequent sore throat. Operation: Local tonsillectomy with no bleeding and no untoward symptoms. The anesthetic used was a 1 per cent solution of novocain and adrenalin prepared in carpules.

On the third day postoperative, the patient started complaining of a pain in the left side of her head and neck. Nothing was found in the throat, and an internist examined her and diagnosed a cervical myositis. On the fifth day the patient had a severe

chill, followed by a rise in temperature to 105. At this time there was a little bogginess of the neck on the left side at the angle of the jaw, and also a slight swelling at the upper pole of the left tonsillar fossa, which was incised and no pus found. The patient was admitted to the hospital for observation.

Blood count, 19,000, with 92 per cent polymorphonuclears. Urine, slight albumin, few hyaline and granular casts. Blood culture negative.

There was an extreme toxemia, and stimulation and sustaining measures were necessary.

On the seventh day a bogginess appeared on the left posterior pharyngeal wall. This was incised and there was a discharge of foul, grayish colored pus and gas bubbles, which on culture proved to be a mixture of the staphylococcus albus and the bacillus proteus vulgaris. The temperature during this time was typically septic.

On the eighth day there was a sudden profuse hemorrhage, which was controlled by pressure after the patient had lost a good deal of blood. She was put on the operating table immediately on my arrival at the hospital, which was about twenty minutes after she started to bleed. There was no further bleeding, but it was decided to tie off the carotid artery to prevent a recurrence. While this was being done she was receiving a saline hyperdermoclysis and stimulation. The tissues separated, as we dissected out the vessel, had the typical grayish, necrotic appearance, and there was an escape of gas bubbles with a very offensive odor. As we finished tying off the vessel, the patient gasped and ceased breathing. She did not respond to any attempts at resuscitation. No anesthesia had been given. Apparently the shock of the hemorrhage added to the extreme toxemia had been too much for her. Further examination of the tissues of the neck at this time showed a generalized involvement by this same putrefactive agent.

Case 3.—Male, 55 years of age, heavily built. He had a chronic myocarditis, with a systolic blood pressure of 180, diastolic 100. His tonsils were large, cryptic and diseased, and he gave a history of repeated sore throats, with rheumatic involvement.

Because of his poor general condition, it was decided to forego any operative measures and to use radium emanation seed implants. Two platinum seeds were implanted, one into each tonsil. This was done in the usual manner, with the use of a large needle and stylet provided for that purpose, and the seeds were placed in the tonsils proper.

On the third day the patient complained of an extreme sore throat, chilliness and fever, which were due to a follicular tonsillitis. The seeds were removed and a gargle and a salicylate prescribed. On the fifth day the temperature was down to normal, the follicles had cleared up, and the patient felt very much relieved. On the sixth day the temperature went up to 103, and the patient again complained of an inability to swallow. At this time there was a peritonsillar swelling on the left side, in which there was no pus on exploration. One day later there was a noticeable swelling on the outside of the neck on the same left side. He became very restless during the night, had a chill, his temperature went up to 105, and he was rushed to a hospital. An incision was made externally and no pus was found, but there was an escape of gas bubbles with a very offensive odor. A severe hemorrhage from the throat followed, apparently from the left tonsillar area, and he died before anything could be done. In this case the myocardium gave way before the process could go any further. There was no culture, but the picture is one of proteus bacillus vulgaris infection.

Comment.—The bacillus proteus vulgaris has been isolated and cultured by M. P. Larson in twenty-three different and distinct strains. They were injected into guinea pigs and rabbits, and showed a range in pathologic changes from the extremely virulent to the very mild.

It is a Gram negative, actively motile bacillus, resembling the colon-typhoid group. It liquefies gelatin in 36 to 48 hours, producing a foul gas, and on being grown and transplanted rapidly loses its toxicity. Pathologically it produces a putrefactive necrosis. The symptoms are essentially an extreme toxicity with an extraordinary loss of body weight. The bacillus occurs ordinarily in the gastrointestinal tract.

According to Martin W. Ware, this organism becomes virulent in tissues that have been prepared by an invasion of another organism. It then takes hold and there is the progressive putrefactive necrotic process, which usually terminates fatally for the host because of the extreme toxemia. In the last two cases death was hastened by hemorrhage with the attendant shock.

In the first case, because of its location, the wound was laid wide open and there was constant drainage. The use of permanganate helped to introduce oxygen, which would inhibit the growth of an anaerobe. We were rather fortunate to be able to stop the process in view of the exposed sinus. On two different occasions, the extreme toxemia nearly overwhelmed the patient. During the prolonged treatment there was a noticeable lessening in intensity of the odor, the organism apparently changing its strain. There have been few reports of infection of the ear area by this organism. Olmacher reports a chronic ear infection which involved the cerebrum and cerebellum. This case terminated fatally and the autopsy showed the characteristic putrid destruction, with the offensive gas and pus formation.

In case 2, there was absolutely no contraindication to operation, and there had been no recent sore throat. The ampoules used were cultured and found sterile. Apparently there was a deep seated focus which was lit up as a result of the trauma of the operation, and the ever present proteus bacillus had a very fine culture medium in the deep tissues of the neck to develop in. One is led to wonder that this complication does not occur more often.

In case 3, we have a similar set of circumstances as in case 2. The reaction to the seeds ordinarily is very mild, but here also there was a deep seated focus, which was lit up by the slight trauma, and the proteus infection was extremely virulent. The woman was younger and stronger, and was able to withstand a good deal more than the older man, who already had the weakened myocardium.

CONCLUSIONS.

1. Bacillus proteus vulgaris infection occurs in the ears and throat, as well as elsewhere in the body.

2. The characteristics are a slow, progressive putrefactive necrosis, with extreme toxemia and an extraordinary loss of body weight.

3. The treatment should be to get at the infected area early and to expose it widely, cleansing frequently and using antiseptics rich in oxygen.

4. The prognosis depends a good deal on the site of invasion, the virulence of the organism and the individual resistance. If drainage is adequate and the resistance is kept up, there is a possibility of recovery, due to the character of the organism, which has a tendency to diminish in toxicity as time goes on.

5. The infection is usually a terminal one. It is rather surprising that one does not encounter this infection more often, in view of the fact that one is always operating through an infected field in tonsil work. The only explanation is that there must be present the peculiar circumstances that were present in these cases and that the proteus bacillus be of a particularly virulent strain.

225 East Nineteenth Street.

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LXXXIV.

FOREIGN BODY IN THE PERITONSILLAR REGION.*

ALEXANDER F. LASZLO, M. D.,

NEW YORK.

The peritonsillar abscess is a very common occurrence in nose and throat practice. With regard to its evacuation opinion is divided between the use of sharp or dull instruments and between wide incisions or puncture. There are very few who advocate packing the abscess after the incision has been made. Patients packed overnight suffer severe pain before the packing is removed.

I am reporting herein a case in which the packing was left *in situ*, either through the forgetfulness of the physician, or the failure of the patient to call at the doctor's office after the opening had been made. The cause of the failure to remove the drain was not determined at the operation.

Z. A., 34-year-old male patient, came to the Nose and Throat Clinic of the Post Graduate Hospital, in June, 1929, with the following complaints: He had suffered from a sore throat many times previously. In January, 1929, he had quinsy on the left side, which had been opened twice by the family physician. When the second opening was made, the physician had inserted some rubber tissue drain. The patient recovered shortly after, but felt a steady irritation on the left side of the throat. He had not complained of any actual soreness of the throat since his previous illness. On June 5th, 1929, he came to the clinic with the complaint of irritation of the throat.

The examining physician found the left tonsil markedly swollen; harder in consistency than the right. No soreness and no inflammatory reaction was present at the time. He advised him to have his tonsils removed. At the operation, the separation of the tonsil on the left side was quite difficult, owing to the many adhesions which were attributed to the previous quinsy; above the upper poles there was quite a large cavity containing two pieces of rubber drain, which were removed.

After removal of the drain, the tonsil was taken out with considerable difficulty, due to the adhesions. There was very little bleeding. The patient went home immediately following the operation, and made an uneventful recovery.

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*From the Nose and Throat Department of the New York Post Graduate Hospital and Medical School.

The two pieces of rubber tissue drain were about 8 cm. long and 2 cm. wide.

Aside from the unusualness of the case, it is interesting to note that two such large foreign bodies can remain in the peritonsillar area, without causing more complaint and without breaking down.

124 East 84th Street.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in Chicago, October 27, 1930, during the session of the American Academy of Ophthalmology and Otolaryngology.

One hundred and eleven candidates were examined, of which eighteen were either conditioned or failed.

The Board will hold an examination in Los Angeles in the spring, preceding the meeting of the Pacific Coast Oto-Ophthalmological Society, and in Philadelphia at the time of the meeting of the American Medical Association. An examination will also be held in the fall, at Indianapolis, preceding the meeting of the American Academy of Ophthalmology and Otolaryngology, which will be held at French Lick Springs.

Prospective applicants for certificates should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Bldg., Omaha, Nebraska, for proper application blanks.

Books Received.

Diathermy, Medical and Surgical, in Otolaryngology.

By Dan McKenzie, M. D., F. R. C. S. E., Consulting Surgeon, Central London Throat and Ear Hospital; Oto-Laryngologist to the French Hospital, London. A volume in the Anglo-French Library of Medical and Biological Science, edited by F. G. Crookshank, M. D., F. R. C. P. (London), and René Cruchet (Professor in University of Bordeaux). Cloth. 8vo of 184 pages with 23 illustrations. New York: The MacMillan Company, 1930. Price, $4.00.

This little handbook is a complete and simple treatise upon the nature of the high frequency currents generating heat in the tissues and their practical application to otolaryngology.

New principles in the treatment of disease have often to live down the fulsome praises of men of little reputation and questionable judgment; so it has been with diathermy. The glowing reports which it has occasioned have so often emanated from such sources that a not unwarranted wave of sentiment occurred against it. Such works as this one by McKenzie serve a useful purpose in putting the matter in its proper light and pointing out in an unbiased fashion the indications and contraindications for its use.

The book is well and profusely illustrated.

Cancer of the Larynx.

By Sir St. Clair Thomson, M. D., F. R. C. P., F. R. C. S., Emeritus Professor of Laryngology in King's College Hospital and Lionel Colledge, M. B., F. R. C. S., Surgeon to the Throat Department, St. George's Hospital, London. A volume in the Anglo-French Library of Medical and Biological Science. Edited by F. G. Crookshank, M. D., F. R. C. P. (London), and René Cruchet (Professor in the University of Bordeaux). Cloth. 8vo of 244 pages with 106 illustrations. New York: The MacMillan Company, 1930. Price, $7.00.

It is rarely that a book comes to hand as satisfying as this one of Sir St. Clair Thomson and Lionel Colledge. According to their own statement the work has been prompted partly by the discouraging dicta of the representative authors of the last century, who characterized the disease as incurable and put it down as a "dire" and hopeless malady; partly by the prevailing enthusiasm of the moment for radium which, according to the authors,

has obscured the achievements of surgery without replacing them. Reference is made to the work of Butlin and other British authors and the advancement in the treatment of laryngeal cancer which followed the establishment of laryngofissure as the route of approach in certain cases.

"The moment appears propitious," say the authors, "for writing a sequel to Butlin's essay of forty-six years ago. It is based partly on opportunities for seeing and appreciating what has been done elsewhere, but chiefly on personal work and experience."

The work is characterized by thoroughness and completeness. The illustrations alone are worth the price of the book.

Diseases of the Ear.

By Philip D. Kerrison, M. D., Consulting Aural Surgeon to the Manhattan Eye, Ear and Throat Hospital and to the Willard Parker Hospital for Infectious Diseases. Cloth. 8vo of 627 pages with 332 illustrations in text and two full pages in color. Philadelphia: The J. B. Lippincott Company, Fourth Edition—Revised and Enlarged, 1930.

No one beyond the third year medical student will require an introduction to Kerrison's outstanding text on the diseases of the ear. The present fourth edition is essentially a revision of the other three, which in itself is as high praise as a book could receive. It is recommended to all students of general medicine as well as those specializing in otology.

The Mechanism of the Larynx.

By V. E. Negus, M. S., London, F. R. C. S., England, Junior Surgeon for Diseases of the Throat and Nose, King's College Hospital, London. Late Arris and Gale Lecturer, Hunterian Professor, and Hunterian Medalist, Royal College of Surgeons of England. Cloth. 8vo of 528 pages with 160 illustrations. St. Louis: The C. V. Mosby Company, 1930. Price, $16.50.

The most complete work written on the comparative anatomy and physiology of the larynx. It contains studies of the structural characteristics, the position and the function of the larynges of every conceivable type of vertebrate. To review this work work adequately would require almost as much space as the work itself, since it delves into innumerable ramifications of the function of the upper respiratory tract.

The author has interpreted the fundamental function of the larynx to be the protection of the pulmonary tract against the invasion of anything beside air. He points out that laryngeal

development is not paralleled by voice production and that the proper use of the pectoral muscles depends largely upon the fixation of the air column by the larynx. The following chapter headings give some idea of the scope of this book:

Chapter I. Introduction.
Chapter II. Evolution of the Larynx.
Chapter III. Modification for Olfaction.
Chapter IV. Modifications for Respiration.
Chapter V. Modifications for Specialized Mechanisms of Respiration.
Chapter VI. Function of Movements at the Glottis During Respiration.
Chapter VII. Modifications for Deglution.
Chapter VIII. Modifications for Regulation of Intrathoracic Pressure.
Chapter IX. Purposive Use of Sound in Relation to the Sense of Hearing.
Chapter X. Employment of Sound as a Means of Communication.
Chapter XI. Mechanism of Phonation.
Chapter XII. Physiological Anatomy of the Human Larynx.
Appendix I. Observations on the Evolution of Man from the Evidence of the Larynx.
Appendix II. Tabulation of Habits of Animals, Together with Anatomical Structure of Upper Air Tract.

A monumental work.

Oral Diseases.

By James L. Zemsky, D. D. S., Attending Oral Surgeon to the Midtown Hospital, New York; Lecturer on Oral Surgery, Post-Graduate Courses, Allied Dental Council; Member Fédération Dentaire Internationale. Cloth. 8vo of 402 pages with 414 illustrations. Brooklyn: Physicians and Surgeons Book Co., 1930.

This book is remarkable for its clarity of exposition and its peculiarly readable style. The paragraphs, which are individually numbered, are short and to the point, each one a definite statement and practically every one illustrated.

Abstracts of Current Articles.

Mixed Chancre of the Tonsil (Le Chancre Mixte de l'amygdale).

Vialle, LeCocq & Ronchèse (Nice). Arch. Int. Lar., 9:513, May, 1930.

These authors report two cases in which Vincent's spirillum and the fusiform bacillus attacked syphilitic lesions of the tonsil, with lesions simulating neoplasms and of extreme discomfort and danger to the patient. They suggest the desirability of tests for lues in all ulcerative lesions of the tonsil, no matter what the smear or dark field may show. F.

Primary Malignant Tumors of the Ethmoid (Tumores Malignos Primitivos del Etmoides).

J. M. Barajas y de Vilches (Madrid). Rev. Esp. Am. de Lar. Ot. Rin., 21:193, May, 1930.

Biopsy and radiographic evidence of bone destruction are invaluable aids in diagnosis. Unless complete surgical extirpation is possible, he recommends radiotherapy and electrocoagulation after broad external access has been gained. Sarcomata are most sensitive to radioactivity, while prickle-celled and dermoid carcinomata are most difficult both for rays and for surgery. He cites two fatal cases in which insufficient radiation was given. F.

Clinical Value of Pulsation of the Lateral Sinus (Valore Clinica della Pulsazione del Seno Trasverso).

S. Valenti (Florence). Bull. della Mal. dell'Oresch., Gol. Naso, 48:129, May, 1930.

Based on the extensive material of Torrini's clinic, the following conclusions are reached:

1. The transverse sinus normally does not pulsate.

2. When, as in cases of thrombosis, pathologic alteration abolishes or greatly reduces its elasticity, pulsation may occur; generally this represents an extensive change within the vessel (complete thrombosis), more rarely a partial alteration (mural thrombosis).

3. In certain cases with a patent lumen there may be apparent pulsation due to periphlebitic changes strongly compressing the

sinus in its bony groove. When such causes are removed, the pulsation stops.

4. The pulsating movement is due to transmission of pulsation of the brain.

5. A thrombosed lateral sinus may fail to pulsate occasionally; in all probability such cases are due to purulent softening of the thrombotic mass.

A complete résumé of various theories precedes the author's review of 173 cases. F.

The March (1930) number of the Archivio Italiano di Otologia, Rinologia e Laringologie is a memorial to Professor Gradenigo (died 1926), containing a picture of the bronze bust recently dedicated to him in the Surgical Aula of the University of Turin, with numerous laudatory addresses and a bibliography of his 355 titles.

Subscriptions for a bust of the late Professor Gherardo Ferreri of Rome are now being received by the Editor (Dr. A. Orlandini), Bolletino delle Oplattie dell'orecchio, della Gola, del Naso, 1 Piazza Madonna, Florence, Italy.

Professor Deodato de Carli, successor to Professor Gradenigo in Naples and as editor of the Italian Archivi, died in June, 1930.

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